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SYMPTOMATOLOGY OF SUBDURAL HEMATOMA IN INFANTS AND IN ADULTS

Comparative Study, with Particular Reference to the Ocular Signs; an Observation Concerning Pathogenesis of Subdural Hematoma

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AND

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BALTIMORE

THE SYMPTOMS of subdural hematoma in adults are quite different from those which occur in infants. The differences in the ocular signs in patients of these age groups suggested this study. Postmortem examination of infant and adult brains led to an observation which seems to explain important differences in symptomatology.

The first part of this paper concerns symptoms and is based on personal observations and studies on 37 infants and 54 adults. Particular attention has been given to differences in symptoms, both general and ocular, in infants and in adults. The second part records an anatomic observation which may explain differences in manifestations in the two age groups.

TERMINOLOGY

Subdural Hematoma.—This term describes any collection of blood in the subdural space, whether or not it is encapsulated. Obviously, in cases in which operation is undertaken immediately after an injury there has not been sufficient time for encapsulation to occur. Little is added by subdividing subdural hematomas into acute and chronic types.

Subdural Hydroma.—This term is applied to subdural accumulations of fluid which may or may not contain a small amount of blood. If the amount of blood present determines the connotation of "subdural hematoma" or "subdural hydroma," it is obvious that in many instances either term would be proper. However, subdural hydroma may result not only from trauma (the usual cause of subdural hematoma) but also from extradural infections, sinus thrombosis, meningitis and hydrocephalus; consequently, the character of the subdural fluid may vary within wide limits. Usually it has a higher protein content than the

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spinal fluid. In some instances the fluid becomes infected and an abscess develops. The nature of the fluid of a subdural hydroma, therefore, varies with the etiologic factor involved.

Pachymeningitis Haemorrhagica Interna.—This term was used by earlier clinicians to describe what is now usually designated as subdural hematoma. Trotter,[1] as long ago as 1914, pointed to the inaccuracy of the term. In certain cases to which the term "pachymeningitis haemorrhagica interna" might properly be applied the condition is, in our opinion, accurately described as "subdural hydroma."

ETIOLOGY AND SYMPTOMATOLOGY OF SUBDURAL HEMATOMA IN INFANTS AND IN ADULTS

Occurrence.—Subdural hematoma is a common occurrence in infants. In our series there were 36 infants and only 1 older child, 8 years of age. Of the 36 infants, 5 were premature. The adult group comprised

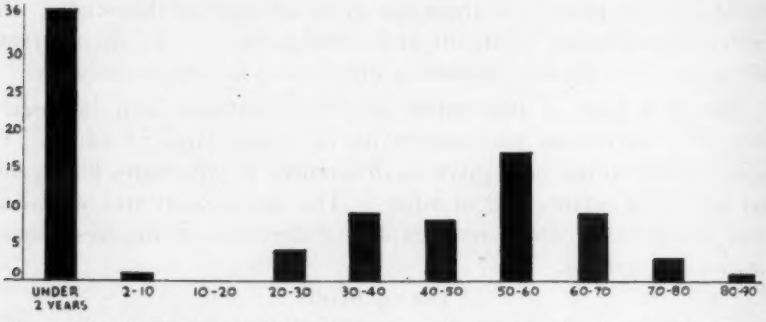

Fig. 1.—Age incidence of subdural hematoma in a series of infants and adults.

54 patients. It is noteworthy that the condition occurred most frequently before 2 years of age and again in the third, fourth, fifth, sixth and seventh decades of life. We had only 1 patient in the age group from 2 to 20 years.

Sex.—Of 36 infants 21 were males and 15 females. Of the adults 48 were men and 6 women. The sex distribution was notably more nearly equal in infants, in contrast to adults, in whom males predominated.

Trauma.—Nineteen of the 37 infants (1 child) and 49 of the 54 adults had a history of trauma. It is more difficult to assess the importance of trauma in infants than in adults. Because of the elasticity of the skull in infants, fractures are not common.

Nutrition.—Of 35 infants, the general state of the nutrition was poor in 14 and good in 21. In the case of 1 infant no record of the

1. Trotter, W.: Chronic Subdural Hemorrhage of Traumatic Origin, and Its Relation to Pachymeningitis Haemorrhagica Interna, Brit. J. Surg. **2**:271-291, 1914-1915.

nutritional state was given. In the adult group we were unable to assess accurately the state of nutrition. Scurvy was clinically obvious in 4 infants and rickets in 3, and both conditions were present in another infant. With regard to the vitamin deficiency status of the infants and

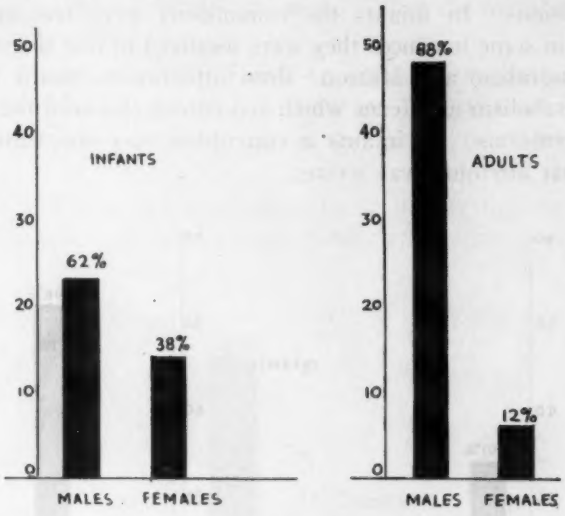

Fig. 2.—Sex incidence of subdural hematoma in 36 infants and 54 adults.

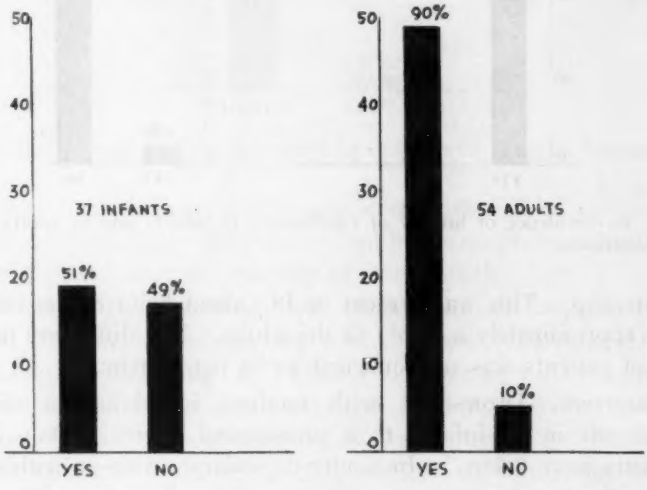

Fig. 3.—Incidence of history of trauma in 37 infants and in 54 adults with subdural hematoma.

adults, our records have little or no value. However, examination of sections of bone taken from 14 infants who died as a result of subdural hematoma showed that the changes in bone characteristic of scurvy

were present in 10.[2] Among the poorly nourished infants evidence of infection of one type or another was frequently present.

Convulsions.—Convulsions were present in 28 infants (and absent in only 7). This is in contrast to the adult group, only 2 of whom had convulsions. In infants the convulsions were frequently generalized, but in some instances they were localized to one side of the body or face. Ingraham and Matson [3] drew attention to tetany due to low calcium metabolism and fever which sometimes obscured the true cause of the convulsions. In infants a convulsion was frequently the first evidence that anything was wrong.

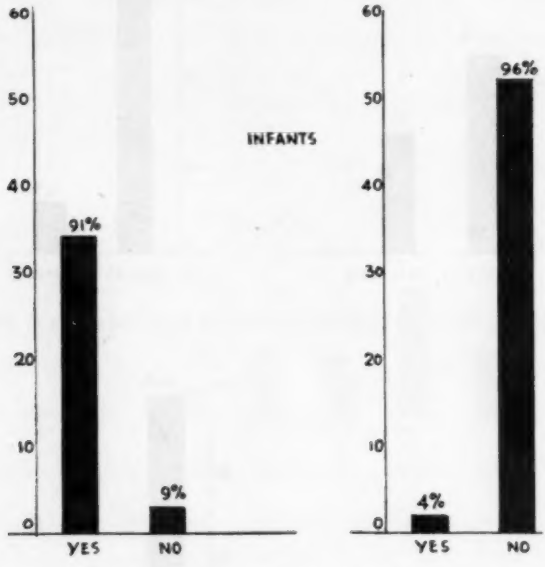

Fig. 4.—Incidence of history of convulsions in infants and in adults with subdural hematoma.

Vomiting.—This was present in 18 (about half) of the infants and in 16 (approximately a third) of the adults. The difference in the two series of patients was not sufficient to be noteworthy.

Sensorium.—Drowsiness with retained irritability to stimulation was present in 20 infants to a pronounced degree. Only 2 of the 36 infants were "alert." In adults drowsiness, with or without irritability, was an extremely common symptom. The drowsiness merged

2. The sections of bones were reviewed by the late Dr. Samuel Blackman, of the department of pathology of the Johns Hopkins University.

3. Ingraham, F. D., and Matson, D. D.: Subdural Hematoma in Infancy, J. Pediat. **24**:1-37, 1944.

into coma in a number of cases. Nine infants (one fourth) and 19 adults (roughly one third) were comatose when first examined.

Bulging Fontanel and Enlargement of the Head in the Infant.—Bulging of the fontanel was exceedingly common; 26 of the 28 infants with an open fontanel exhibited the sign. When the fontanel was open, this sign was practically constant.

Fever.—Twelve of the 36 infants on admission showed an elevation of temperature to 38 C. (100.4 F.) or above. We do not have accurate figures on the adult patients, but in the acute stages fever may be expected.

Neurologic Signs.—In many adults and infants definite neurologic signs were absent. As already mentioned, a common feature in most

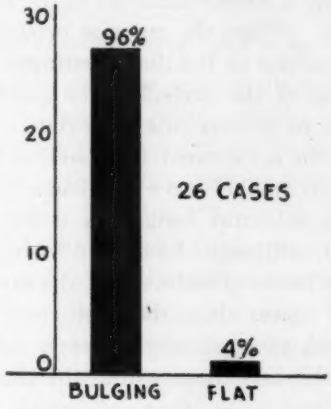

Fig. 5.—Incidence of bulging fontanel in 28 infants with subdural hematoma.

patients was increased irritability and drowsiness. The ocular signs of importance—pupillary dilatation, retinal hemorrhages and papilledema—and their significance are considered subsequently.

In 51 adults the hematoma was unilateral, and hemiparesis was present in 22. In 17 patients the weakness was contralateral to the lesion, and in 4, ipsilateral. In 1 patient hemiparesis was present when the hematoma was bilateral. Hemiplegia ipsilateral to the hematoma is explained on the basis of pressure of the tentorium on the opposite side of the displaced brain stem. The hemiparesis was associated with tendon hyperreflexia, the Babinski sign and ankle clonus. The hemiparesis usually developed late, was rarely complete and varied in severity from time to time. In a few instances the paresis was flaccid.

In infants, from the standpoint of diagnosis, the neurologic signs were of secondary importance to bulging of the fontanel, blood in the

spinal fluid and drowsiness. However, in their essential features they were the same as in adults except that the signs were usually bilateral.

Spinal Fluid.—In infants the spinal fluid invariably contained blood or was xanthochromic. In the great majority of adults the spinal fluid was normal.

OCULAR SIGNS OF SUBDURAL HEMATOMA

Subdural Hematoma of the Optic Nerve Sheaths.—This condition, although not described here in any detail, deserves mention. A brief statement regarding the disposition of the dura mater in the optic canal and orbit is necessary to an understanding of it. The dura mater, which surrounds the brain and is attached to the skull, is continuous about the optic nerve, which it reaches at the entrance to the optic canal. As the optic nerve passes toward the eye through the canal, it is surrounded by dura, which is loosely attached to the bony walls of the canal and to the optic nerve. When the anterior orbital opening of the canal is reached, the inner portion of the dura continues about the optic nerve to the posterior surface of the eyeball. The outer portion of the dura leaves the optic canal to become the periorbita. Because there is no real subdural space in the optic canal, it is obvious that a subdural hematoma within the orbit is unlikely to extend into the cranium. Also, it seems unlikely that a subdural hematoma within the cranium would extend into the orbit, although Liebreicht [4] suggested that subdural hematoma of the optic nerve sheaths might develop as a result of blood passing along the fluid spaces about the ophthalmic artery into the orbit. He demonstrated a lack of continuity between subdural accumulations of blood within the orbit and those within the cranial cavity.

We are unable to state precisely the ocular signs of orbital subdural hematoma because a great majority of persons who have such a lesion exhibit signs which are in general attributed to injury but are not considered as indicating operation. Our clinical studies point to absence of orbital subdural hematoma in cases of intracranial subdural hematoma. It is of interest that subarachnoid hemorrhage within the cranium is frequently associated with a similar accumulation of blood within the orbit. This is in sharp contrast to what prevails with subdural hematoma. Probably subdural hematoma of the optic nerve sheaths occurs not uncommonly with subarachnoid hemorrhage, both intracranial and intraorbital.

Intracranial Subdural Hematoma.—The ocular signs in infants and in adults are quite different, and the differences are important.

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4. Liebreicht, cited by Cone, W., and MacMillan, J. A., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 2, pp. 848-850.

Papilledema.—The occurrence of papilledema in adult patients has been estimated by several observers to be about 50 per cent (Dandy,[5] Kunkel and Dandy,[6] King,[7] Furlow [8]). This figure seems high on the basis of our observations. It is also high according to the case records we have used in the preparation of this paper. We found mention of papilledema in approximately 17 per cent of both the adult and the infant patients.

The papilledema seen in adults is precisely similar to that which is observed in many cases of cerebral tumor; that is, there are varying amounts of swelling of the optic disks, and there may or there may not be retinal hemorrhages of nerve fiber layer type. The papilledema which is sometimes seen in infants is, according to our experience, almost invariably associated with subhyaloid retinal hemorrhages. In both infants and adults, long-standing papilledema is likely to be associated with optic atrophy of the nerve and loss of visual acuity. It is our belief that papilledema is a late development in these patients.

Retinal Hemorrhages.—In the series here reported the frequency of subhyaloid (preretinal) hemorrhages in infants and their uniform absence in adults are both noteworthy and difficult to explain. In this regard, it is again remarked that in infants there usually is blood in the spinal fluid (or xanthochromia) and that the spinal fluid in adults is clear. It is generally believed that subhyaloid bleeding is venous in origin, but the exact mechanism responsible for the production of preretinal hemorrhage is unknown. Perhaps there are episodes of relatively greater and more sudden increases in intracranial pressure in infants than in adults. We have come to believe that in a healthy infant the presence of subhyaloid (preretinal) hemorrhage almost certainly indicates a subdural hematoma. In an adult the presence of such a hemorrhage almost rules out subdural hematoma.

Pupillary Signs.—Careful observation of the state of the pupils is of the greatest possible importance, particularly in adults. It is of less significance in infants because in them the lesion usually is bilateral. In adults one pupil may be dilated and fixed, or relatively fixed, to light on the side of the lesion. Infrequently the pupil contralateral to the lesion is dilated. Dilatation of the pupil may be transitory and

5. Dandy, W. E.: Subdural Hematoma, in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1932, vol. 12, pp. 848-850.

6. Kunkel, P. A., and Dandy, W. E.: Subdural Hematoma: Diagnosis and Treatment, Arch. Surg. **38**:24-54 (Jan.) 1939.

7 King, C.: Chronic Traumatic Subdural Hematoma as a Cause of Choked Disc, Tr. Am. Ophth. Soc. **63**:732-742, 1936.

8. Furlow, L. T.: Chronic Subdural Hematoma, Arch. Surg. **32**:688-708 (April) 1936.

slight. Such dilatation, according to Woodhall and his associates,[9] is as significant from the viewpoint of localization as more prolonged dilatation.

We are in complete agreement that unilateral dilatation usually signifies the side of the hematoma. However, in the series of cases on which we have based this communication pupillary dilatation could not be considered an important sign. Possibly in some instances transient dilatation was overlooked. The pupil was dilated and fixed or sluggish in its reaction to light on the side of the lesion in 5 adult patients, and in 1 patient the pupillary change was contralateral to the lesion. These figures are small as compared with those of Kennedy and Wortis,[10] who found in a series of 72 cases of subdural hematoma homolateral

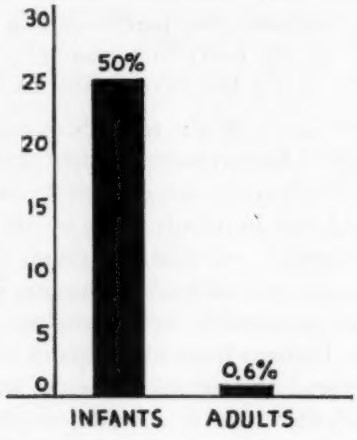

Fig. 6.—Incidence of retinal hemorrhages in infants and in adults with subdural hematoma.

dilatation of the pupil in 30 and contralateral dilatation in 12. Kunkel and Dandy gave this sign little consideration in their study. In 1 adult with bilateral subdural hematoma we observed unilateral pupillary dilatation.

There is general agreement, and we have observed, that dilatation of the pupil disappears certainly within a few hours, and sometimes within a few minutes, after evacuation of the hematoma.

It is only recently that we have had an explanation for homolateral dilatation and fixity of the pupil in cases of subdural hematoma and of

9. Woodhall, B.; Devine, J. W., Jr., and Hart, D.: Homolateral Dilatation of Pupil, Homolateral Paresis and Bilateral Muscular Rigidity in Diagnosis of Extradural Hemorrhage, Surg., Gynec. & Obst. **72**:391-398, 1941.

10. Kennedy, F., and Wortis, H.: Acute Subdural Hematoma and Acute Epidural Hemorrhage, Surg., Gynec. & Obst. **63**:732-742, 1936.

subarachnoid and extradural hemorrhage. Reid and Cone [11] and Woodhall and associates, among others, described herniation of the hippocampus or the medial aspect of the temporal lobe and midbrain through the tentorium. As a result of such herniation, there is pressure on the homolateral third nerve, with the development of an internal ophthalmoplegia, which may be associated with ptosis. Reid and Cone demonstrated such herniation in experimental animals and found that homolateral pupillary dilatation developed. In their animals there were no hemorrhages in the midbrain, but in some instances hemorrhages developed in the occipital lobes. Although absence of hemorrhages in the midbrain suggests that involvement of the nucleus of the third nerve is not responsible for the pupillary dilatation, the experimental work of Magoun and associates [12] is even more convincing in this regard. These observers found that stimulation of the gray matter in the region of the nucleus invariably produced bilateral pupillary constriction, whereas stimulation of the peripheral nerve invariably produced homolateral pupillary narrowing. Ecker and Anthony [13] suggested that traction of the third nerve on the sphenoid bone, rather than pressure on the nerve, may be the cause of the pupillary change. Although there can be no reasonable doubt that herniation occurs, as has been described, and that it is associated with the pupillary change, the actual mechanism is not completely understood. The pupillary pathways are known to be peripheral in the nerve. While this explains the internal ophthalmoplegia, it is difficult to understand why a more complete paralysis of the third nerve does not occur frequently. We have never observed total paralysis of the third nerve in a case of subdural hematoma but have observed such a paralysis frequently as a result of aneurysm, and in both instances pressure on the third nerve seemed to be the etiologic factor.

Both in infants and in adults our series contained cases of bilaterally dilated and fixed pupils. Possibly hemorrhages in the midbrain were present in these cases. Also, we observed bilateral narrowing of the pupils, which seemingly was an expression of an irritative miosis.

Exophthalmos.—We observed bilateral proptosis in 1 infant not included in this series, but there was free blood in the orbital cavity.

Ptosis.—Ptosis may or may not be present on the side of pupillary dilatation. Unilateral ptosis was present in 6 patients, and in 5 it was

11. Reid, W. L., and Cone, W. V.: The Mechanism of Fixed Dilatation of the Pupil Resulting from Ipsilateral Cerebral Compression, J. A. M. A. **112**:2030-2034 (May 20) 1939.

12. Magoun, H. W.; Atlas, D.; Hare, W. K., and Ranson, S. W.: The Afferent Path of the Pupillary Light Reflex in the Monkey, Brain **59**:234-239, 1936.

13. Ecker, A. D., and Anthony, E. W.: Head Injuries from the Ophthalmologist's Viewpoint, Brit. J. Ophth. **29**:43-48, 1945.

on the side opposite the subdural hematoma. Possibly such a contralateral ptosis is supranuclear in origin, although, theoretically, abolition of cortical impulses might result in increased tone in the levator muscle. We have not seen cases in which a diagnosis of supranuclear ptosis could be made with certainty. Pronounced bilateral ptosis was present in a single case.

Involvement of the Third Nerve.—We have not seen an instance of total paralysis of the third nerve. Furthermore, we observed ptosis in association with homolateral pupillary dilatation in only 3 patients.

Involvement of the Sixth Nerve.—Of our adult patients, there was unilateral paresis of the sixth nerve in 3 and bilateral paralysis in 1.

Nystagmus.—This sign was mentioned frequently in the case reports of infants. It may have originated in defective ocular fixation. It was present in only 3 adults. Probably the ocular movements of nystagmus in adults are dependent on palsies of conjugate movements.

Conjugate Deviation of the Eyes.—In 2 adults the eyes were deviated away from the side of the lesion. Since this deviation was persistent, it seemed unlikely that it resulted from an irritative lesion; rather, it appeared to be due to an interruption to the supranuclear pathways in the hemisphere opposite the side of the lesion. Conjugate deviation of the eyes was seen frequently in infants as part of convulsive episodes when they did not have recognizable localizing value.

Visual Field Defects.—These were noted in a minority of the patients. Maltby [14] found evidence of a visual field defect in only 7 of 62 cases (11 per cent). The field defects were homonymous and contralateral to the side of the clot in 5 cases, ipsilateral in 1 case and left sided in a case in which the clot was centrally situated. Maltby expressed the belief that the field defects were due to obstruction of circulation through branches of the posterior cerebral artery as the result of herniation of the temporal lobe through the tentorium. He stated that the field changes had more lateralizing value than the pupillary anomalies already described. We observed homonymous hemianopsia for colors in only 1 patient and contralateral hemianopsia in another.

Also, we observed total blindness and total atrophy of the optic nerve in 2 patients. One patient, a youth 18 years of age, was moribund when he was brought to the hospital; his visual acuity was said to have been very defective; after operation it remained at bare perception of light and was unchanged three years later. The other patient was a man who had been operated on elsewhere; he was completely blind at the time of our examination, but he stated that his vision had been tolerably good before his operation.

14. Maltby, G. L.: Visual Field Changes and Subdural Hematomas, Surg., Gynec. & Obst. **74**:496-498, 1942.

Loss of Corneal Reflex.—This has been described as occurring on the side of the lesion. We do not have accurate information regarding it in cases of subdural hematoma.

AN OBSERVATION CONCERNING PATHOGENESIS

In compiling this report, we were attracted by the rarity of subdural hematoma in the age group from 2 to 20 years; the regularity with which the hemorrhage occurred bilaterally in infants and unilaterally in adults; the regularity with which blood (or xanthochromia) was found in the spinal fluid of infants and its almost uniform absence in adults, and the frequent absence of a history of trauma in infants and the almost invariable history of trauma in the adults.

In an attempt to explain these differences, several infant and adult brains were examined. Gross examination revealed an important anatomic difference between the supporting structures of the pial veins of the infant and those of the adult. These differences, although recognized by anatomists, have never been associated with the pathogenesis of subdural hemorrhage.

To demonstrate these differences, it is necessary to divide the dura a few centimeters from the midline of the convexity. In the infant the dura and the delicate arachnoid membrane are easily separated, and when the dura is retracted toward the midline several large pial veins are seen entering the longitudinal sinus. These vessels are short, nontortuous and without support. Grossly, they appear to lie free in the subdural space for distances varying from a few millimeters to 1 cm. before entering the longitudinal sinus. The veins do not pass directly from their location in the pia into the sinus but follow a diagonal path and enter the sinus at an angle.

In sharp contrast to the condition seen in the infant, the dura and the arachnoid membrane of the adult are firmly bound together in the region of the longitudinal sinus. This attachment of the arachnoid to the dura is formed by masses of fibrous tissue, forming the base of arachnoid villi (pacchionian granulations). These differences are apparent in the drawings made from a specimen of brain from a normal infant and one from an adult.

These fibrous attachments were described by Weed [15] as representing fully developed arachnoid villi. The invasion of the dura by the delicate arachnoid villi occurs in infancy. In infants they are microscopic, offer little support and are not visible. Our observations indicate that these structures become visible soon after the period of infancy. The villi observed in 4 and 5 year old children were comparable in development to those of an adult.

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15. Weed, L. H.: Studies on Cerebrospinal Fluid, J. M. Research **26**:51-113, 1914-1915.

The effect of these anatomic differences on the movement of the brain within the cranial cavity is not known. One would think, however, that the movement should be much freer in the infant than in the adult, for the attachment of the arachnoid to the dura in the adult brain ought necessarily to limit its movement. It would seem likely, also, that in the infant when the pial vessels entering the longitudinal sinus

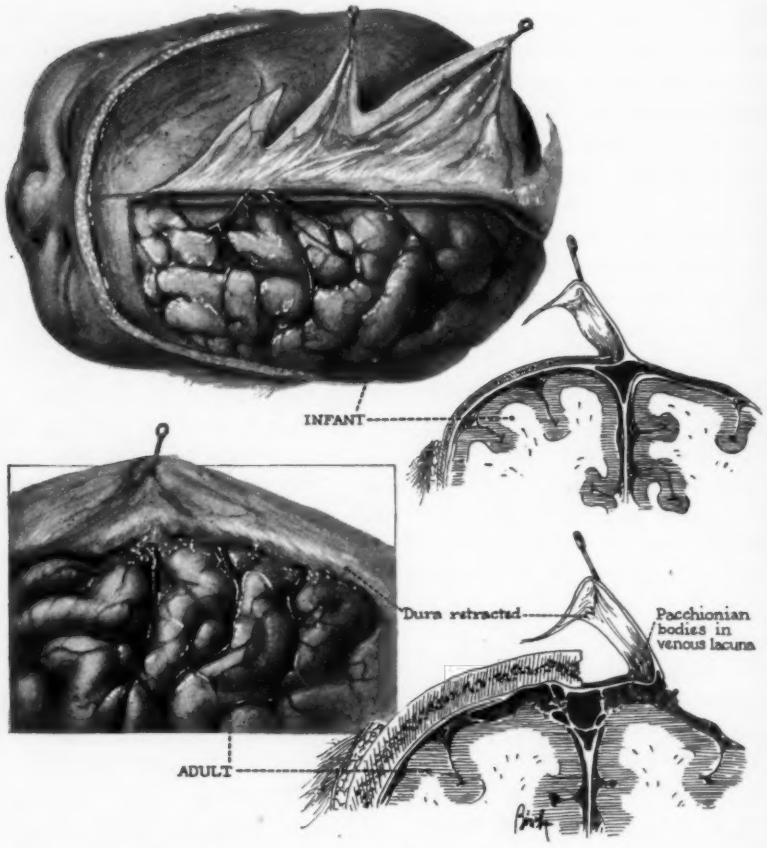

Fig. 7.—Differences in the relation of the dura and the arachnoid membrane in the region of the longitudinal sinus in the infant and in the adult.

rupture the arachnoid would be torn simultaneously and the blood would find its way into the subarachnoid space as well as between the outer surface of the arachnoid and the dura. It is plausible to think that in the adult, on account of the presence of these fibrous attachments, which must protect the pial veins entering the longitudinal sinus, a severer trauma would be required to rupture the vessels. The usual absence of a history of trauma in infants may perhaps be explained on the

ground that only slight trauma might be required to rupture the veins in the infant on account of the freer mobility of the brain. The freer mobility of the brain might also explain why the subdural hematoma in the infant is almost invariably bilateral.

Differences in the anatomic arrangements of the membranes of the brain in the infant and in the adult require further studies.

Wilmer Ophthalmological Institute.

DISCUSSION

Dr. J. Parsons Schaeffer, Philadelphia: I should like to know how many of the subdural hematomas at the early ages mentioned were the result of birth injuries. My other point has to do with the variation in size of the optic foramen through which the optic nerve passes into the orbit. When the opening is roomy, it might be inferred that the subdural hemorrhage would readily pass into the dural-arachnoid interspace and that failure of blood to do so might be due to the bony encroachment on the optic nerve and its sheath and interspaces.

Dr. Francis Heed Adler, Philadelphia: The high incidence of subdural hematoma in infants could be logically explained by the prevalence of birth injuries. Every normal birth exposes the child to cranial compression, and since the fontanels are open then and for some time after birth, it is no wonder that there should be a high incidence of intracranial trauma. From the time that the fontanels close, the child is not often exposed to any serious intracranial damage until he reaches the age at which serious accidents are apt to occur. In other words, the falls and bumps of the nursery do not produce intracranial lesions, but industrial accidents often do. It would seem much more logical to assume that this explains the distribution of subdural hematomas according to age than to base this incidence on an anatomic difference in the cranial contents in the infant and in the adult.

Dr. Walsh has asked me to comment on ptosis as a sign of supranuclear paralysis. Since the muscles of the lid have a separate representation in the frontal cortex, it is possible to have a supranuclear paralysis with a lesion situated anywhere from the cortex down to the nucleus. This, of course, is not true of the individual muscles controlling the movements of the eyeball. Furthermore, the fiber tracts for the levator and muscles (the latter is questionable) run together with the fiber tracts for upward and downward movements of the eyeballs in the region of the corpora quadrigemina. Accordingly, lesions in this region cause disturbances of upward and downward movements of the eyes, together with ptosis and tucked lids. A few years ago Collier called attention to ptosis and retraction of the lid as signs of supranuclear paralysis of the third nerve, differentiating these lesions thereby from lesions affecting the third nerve itself or the nucleus. If the lesion is anterior, retraction of the lid is likely to be present, whereas if the lesion is posterior ptosis occurs.

Dr. Walter I. Lillie, Philadelphia: Dr. Walsh is to be congratulated on his presentation of a difficult subject, as the classification of traumatic cerebral changes is not always well differentiated. Clin-

ically, it is often difficult to correlate the multiple, bizarre changes with a single lesion. Although a single large lesion is present, associated small multiple lesions help to explain the syndrome. Dr. Walsh has not explained specifically why a subhyaloid hemorrhage occurs in infants and not in adults. The occurrence of subhyaloid hemorrhages in adults is usually associated with a compression injury to the chest or the neck, but that it may occur from intracranial venous compression is a possibility.

The diagnosis of subdural hematoma is usually supported by an encephalogram, as the variations in the fluid channels reveal the space-taking lesion.

The occurrence of general convulsions in adults should suggest a lesion of the frontal, temporal or occipital lobe. Such patients should have a routine perimetric examination of the fields, as a quadrantal homonymous hemianopsia has often been found a year or two in advance of other definite signs of a space-taking lesion.

DR. HENRY A. SHENKIN, Philadelphia: I should like to comment from the neurosurgeon's point of view. Dr. Walsh made an effort to distinguish between the symptoms of subdural hematoma in the child and those in the adult, and it is true they are quite different. The fact that there are open fontanels in the infant would well account for the difference in symptoms.

In adults papilledema does occur, but my experience is that it rarely is seen in infants. The open fontanels could permit sufficient expansion of the calvaria to absorb the pressure. The presence of convulsions in childhood may well be due to a lowered convulsive threshold, said to be characteristic of infancy. However, the length of time to which the cortex is subjected to irritation may be the convulsive factor. It is known that cerebral tumors of slow growth are the ones most frequently associated with seizures; and, by analogy, subdural hematoma of long duration may be more frequently associated with seizures. The open fontanels, compensating for the increased mass of the hematoma, permit the child to survive for periods far in excess of the usual period in which the life of an adult would be threatened by a chronic subdural hematoma. It is likely that the subdural hematoma of infancy is caused by trauma at birth. The appearance of the lesion and the history of the child's development substantiate this. The neurosurgeon is usually consulted when the child is 6 to 12 months of age. On the other hand, adults usually manifest life-threatening symptoms from chronic subdural hematoma six to twelve weeks after their injury.

Unilateral dilatation of the pupil is more characteristic of acute subdural hematoma than of the chronic form. I believe subdural hematoma of infancy is more nearly analogous to the latter entity than to the former. Indeed, ipsilateral dilatation of the pupil is far more characteristic of the fulminating syndrome of acute epidural hemorrhage than of the more slowly developing acute subdural hemorrhage.

I agree that the results of treatment of subdural hematoma of infancy are poor. I think that this can be explained by the delay in making a proper diagnosis. The growing brain is compressed for undue periods, and often it never recovers sufficiently to develop to its full capacity.

DR. J. PARSONS SCHAEFFER, Philadelphia: I should like to pose a question. There is some thought that the facial nerve may, in some manner, be responsible for the peculiar behavior of the upper eyelid in certain cases. Some of the fibers that arise from the nucleus of the oculomotor nerve descend in the medial longitudinal fasciculus and either terminate about the cells of the nucleus of the facial nerve or join the facial nerve as such, passing in this nerve to the upper part of the orbicularis oculi muscle, thus associating the innervation of the levator palpebrae muscle with that of the orbicularis oculi muscle. Although it is generally accepted that the oculomotor nerve supplies the levator palpebrae muscle and that the orbicularis oculi muscle is supplied by the facial nerve, this intermingling of the fibers of the two nerves may well be variable enough to account for such peculiar manifestations with reference to the action of the upper eyelid. Much more study is needed.

The other point concerns the newborn infant, to whom Dr. Walsh referred. There is no doubt that the veins as they course to enter the superior sagittal sinus are fairly free, but it is equally true that there is a mesothelium covering them that does not add much protection. Later, in the adult, as the cerebral membranes become more pronounced and the interspaces are developed, the parts become matted together near the superior sagittal sinus. Also, the arachnoid and the subarachnoid spaces, in the form of arachnoidal granulations, protrude into the superior sagittal sinus, the endothelium of course being crowded ahead. This adds to further crowding near the venous sinus and encroachment on the cerebral veins as they enter the dural sinus.

DR. FRANK B. WALSH, Baltimore: In reply to Dr. Schaeffer, I may say that in some infants birth injury is a possible explanation for subdural hematoma. It is extremely doubtful whether it was operative in a majority of our infants. We have not made studies on the optic foramens.

Except for some of the literature concerning the joint innervation by the third, seventh and ninth nerves, I confess to knowing nothing. However, I am flattered to have been asked this question by Dr. Schaeffer, despite my inability to answer it.

Dr. Adler's explanation of supranuclear ptosis involving the lid opposite the hematoma is interesting. However, there is no characteristic feature in such a ptosis that would eliminate the possibility of its having been produced by a homolateral incomplete palsy of the third nerve. I was glad attention was drawn to Collier's paper, which I recommend as excellent.

I agree with Dr. Lillie that in these cases there may be multiple lesions. I am unable to explain preretinal hemorrhages seen in cases of subarachnoid hemorrhage. It is thought that such retinal hemorrhages are venous in origin. One would think they might be associated with sudden increase in intracranial pressure. However, Gardner has shown that the intracranial pressure in many cases is not increased.

CHOROIDEREMIA

Report of a Case

BENJAMIN ESTERMAN, M.D.
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THE TERM choroideremia has been used to describe an apparent absence ophthalmoscopically of the entire choroid except in the macular region. Since the first description, in 1872, by Mauthner,[1] 31 cases of a condition described as choroideremia have been published in the literature. These were cited by Nettleship,[2] Wolf [3] and Parker and Fralick.[4] In 1937 Bedell [5] gave an excellent summary of each of the then published cases which he considered instances of choroideremia in the broader sense; the description of these cases does not need to be repeated here. He added 5 cases of his own. Six years earlier, in a discussion on Parker and Fralick's paper, he suggested that choroideremia may appear in more than one form. Since that time Friedman [6] (1940), Scobee [7] (1943), Shapira and Sitney [8] (1943) and Magder [9] (1945) have reported cases.

Friedman's [6] case was that of a man of 45 who had always experienced night blindness and who stated that one uncle had a similar condition. He had myopia of about 6½ D. with corrected vision of 20/30 + in the right eye and 20/50 — in the left eye and concentric contraction of the fields to about 10 or 15 degrees. The disks were normal, as was the retinal circulation. The choroid was absent except for that in the macular regions, some remaining choroidal vessels and a few clumps of pigment.

From the Manhattan Eye, Ear and Throat Hospital; service of Dr. R. Townley Paton.

Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Jan. 21, 1946. A discussion of this paper was published in the August 1946 issue of the ARCHIVES, page 245.

1. Mauthner, L.: Ber. d. naturw.-med. Ver. in Innsbruck **2**:191, 1871.

2. Nettleship, E.: Roy. London Ophth. Hosp. Rep. **17**:373, 1907-1908.

3. Wolf, S.: Choroideremia, Arch. Ophth. **3**:80 (Jan.) 1930.

4. Parker, W. R., and Fralick, F. B.: Choroideremia: Report of a Case, Arch. Ophth. **6**:213 (Aug.) 1931.

5. Bedell, A. J.: Choroideremia, Arch. Ophth. **17**:444 (March) 1937.

6. Friedman, B.: Choroideremia, Arch. Ophth. **23**:1285 (June) 1940.

7. Scobee, R. G.: Am. J. Ophth. **26**:1135, 1943.

8. Shapira, T. M., and Sitney, J. A.: Am. J. Ophth. **26**:182, 1943.

9. Magder, H.: Choroideremia: Report of a Case, Arch. Ophth. **33**:468 (June) 1945.

Scobee [7] reported 2 cases of the condition in brothers, the disease in the older, aged 24, being in an advanced stage, and that in the younger, aged 18, in an early stage. The absence of symptoms in the childhood history of the patient with the more advanced disease, together with the apparent worsening of the subjective and objective findings during the month in which both patients were under observation, led Scobee to agree with Bedell that the condition is not congenital but acquired. The presence of evident degenerative changes in the vitreous (pigment balls; striation of fibrils in the anterior portion of the vitreous) tended further to point to the acquired nature of the disease in these cases.

Shapira and Sitney's case, the second in which the disease had been reported in a female, was that of a woman aged 52 with hypertension and syphilis of the central nervous system, Argyll Robertson pupils, corneal scarring, pigment, posterior corneal deposits, aqueous flare and posterior synechias. The condition was seen by a number of observers, who pronounced it choroideremia, even in the light of Verhoeff's caution regarding confusion with retinal gliosis.

Magder's case was that of a man aged 58 with no family or personal history of ocular disorder except for night blindness, which had always existed but had become worse during the last ten years. He had low myopia, correctible to 20/20 and 20/30, with fields contracted to within 10 degrees of fixation plus a narrow crescentic peripheral area. The choroid was absent in each eye except for the macular region and the extreme periphery. A few remaining choroidal vessels were seen, as were several clumps of pigment.

The condition is always bilateral; occurs almost invariably in males; is usually associated with night blindness and sharply contracted visual fields, but often with relatively unimpaired central vision, and in the majority of instances occurs in persons with myopia of moderate degree. The history of defective vision and visual fields is usually of long standing, going back as far as the patient can remember in most cases, and the process has often remained stationary during the life of the patient. A few cases have been reported in brothers; but in no instance has there been consanguinity in the parents, nor has any authentic case been found of the existence of the same condition in a parent or a grandparent or in offspring.

REPORT OF A CASE

D. H., a white man aged 29, was admitted to the eye clinic of the Manhattan Eye, Ear and Throat Hospital, service of Dr. R. Townley Paton, on March 6, 1945. His chief complaint was night blindness, which had always been present but which he claimed had increased somewhat in the past few years. Dark adaptation had always been difficult, and distant vision had always been poor without glasses. With glasses his central vision had been such as to enable him to do clerical work. Central color vision was not impaired.

The family history was negative for consanguinity and was otherwise irrelevant except that two uncles were said to have had poor vision, the nature and cause of which were unknown. No evidence could be obtained pointing to syphilis in the past history of the patient or of his family; there was no recollection of ocular or cranial injury or of any ocular inflammatory disease.

The physical examination revealed an entirely normal condition as did dental and rhinologic examinations, roentgenologic studies of the chest, urinalysis, the Wassermann test, the Mantoux test and hematologic studies.

Visual acuity was 5/200 in the right eye, improved to 20/200 with —6 D. sph. ⌒ —2 D. cyl., axis 180, and 10/200 in the left eye, improved to 20/30 with —4.50 D. sph. ⌒ —1.25 D. cyl., axis 180. The visual fields were found to be reduced to within 5 degrees of fixation in each eye, using a 10 mm. white stimulus at 330 mm.

The lids, lacrimal apparatus, conjunctivas and corneas were normal; the anterior chambers were of normal depth; the irises were normal; the pupils reacted properly both directly and consensually to light and in accommodation and dilated readily with mydriatics to disclose a clear lens and vitreous. Transillumination was similar to that seen in albinotic eyes. Intraocular tension was soft. All extraocular movements were unimpaired.

Ophthalmoscopic Examination.—Right eye: The disk was of good color with sharp margin and a small physiologic excavation; the lamina cribrosa was not seen. The retinal arteries and veins were of normal caliber and distribution. A pyriform area 3 by 1½ disk diameters in the macular region and a small pink spot between this and the disk were all that remained of the choroid, the rest of the fundus appearing as a faintly stippled white, with a few scattered pigmented areas, as shown in figure 1, and one rather dense area lying in the macular region, and possibly accounting for the fact that central acuity in the right eye could not be improved beyond 20/200. Passing just behind this patch of pigment was a group of five choroidal vessels, which had perforated the sclera, branched and fanned out temporarily to fade away after running behind an ascending branch of the inferior temporal vein. A few other branches of the posterior ciliary vessels were seen perforating the sclera, superior and nasal to the macular region, while two entered the globe about 3 disk diameters temporal to the macula, passed behind a clump of pigment and faded away as they coursed peripherally.

Left Eye: The disk was of good color; but there was a faint blurring of the margins, only a suggestion of a physiologic excavation was evident and no lamina cribrosa was visible. The retinal vessels were normal. A round area, 3 by 3 disk diameters, in the macular region, a small T-shaped area superior and temporal to the disk and a faint spot in the temporal periphery were the sole remnants of the choroid in this eye. The rest of the fundus was a faintly stippled white with a few pigment areas, one of which plainly lay behind a branch of the superior temporal artery and vein. Emanating from the macular regions were a few choroidal vessels, most of which coursed temporally for a short distance and disappeared, while one traveled nasally and upward to lose itself behind a branch of the superior temporal vein. A somewhat unusual feature was the striking difference in macular vision in the two eyes. In spite of the fact that in each eye the choriocapillaris and the pigment epithelium of the macular area were spared, there was ophthalmoscopic evidence of macular degeneration in the right eye. This was probably quite independent of the choroideremia, since it is conceivable that an eye which has more than 6 D. of myopia may show macular changes in its own right.

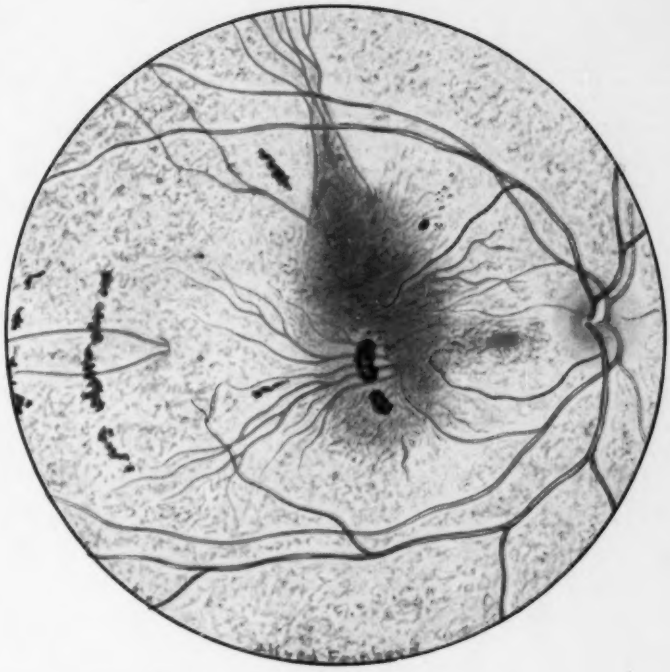

Fig. 1.—Choroideremia, right eye.

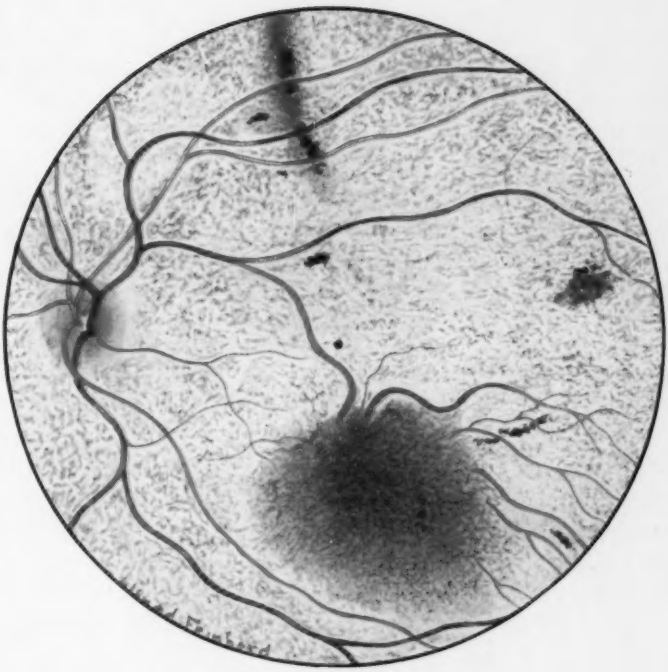

Fig. 2.—Choroideremia, left eye.

COMMENT

The knowledge of the nature and etiology of choroideremia is largely speculative owing to the absence of histologic data. Mann [10] expressed the belief that the defect is due to failure of the posterior ciliary arteries to develop or is associated with failure of the outer layer of the optic cup to produce pigment. She stated:

> The choroidal net seems to develop wherever mesoderm is in contact with pigment epithelium. It appears with the pigment and seems to be in some way related to this, in that if for any reason pigment is absent over an area of the surface of the optic cup, the choroid is absent also.

She cited the instance of a certain area below the optic nerve in birds which is normally unpigmented and over which choroid does not develop. Collins and Mayou attributed the condition to failure in the formation of blood vessels in the inner part of the mesoblast except for the vessels supplying the macula.

In explanation of the sparing of the macular region, Connor [11] said:

> The analog in embryonic life of what is later the macular area of the fundus is a spot in the outer surface of the secondary optic vesicle where the first signs of differentiation of the mesoblast into sclera and choroid are seen, the first appearance of the choroid being manifest by a tiny plexus of capillaries at this point. Hence, in seeking an explanation of the cause of choroideremia, we must consider that differentiation of the mesoblast of the secondary optic vesicles into sclera and choroid began in a normal way and at the normal point, but for some reason was arrested when only the macular region was supplied with choroid.

Nettleship explained the appearance of patches of pigment by the fact "that the retinal epithelium is laid down in the first few weeks of embryonic life before the choroid which is to nourish it has been differentiated; therefore, if the choroid atrophies or does not develop, we should expect that the epithelial pigment, so far as it has been formed, would retrogress and migrate."

DIFFERENTIAL DIAGNOSIS

Choroideremia must be distinguished principally from retinitis pigmentosa, gyrate atrophy, choroidal sclerosis and massive retinal gliosis.

Uncomplicated retinitis pigmentosa is easily differentiated by the presence of many bone-corpuscle-like deposits of pigment, the waxy pallor of the disks and the marked attenuation of the retinal vessels, all of which are absent in choroideremia.

The gyrate atrophy of Fuchs and Cutler characteristically begins in the periphery with numerous irregular areas of choroidal atrophy, separated by normal-appearing fundus. These areas grow larger and often

10. Mann, I.: The Development of the Human Eye, New York, The Macmillan Company, 1928; Developmental Anomalies of the Eye, ibid., 1937.

11. Connor, A. B.: Am. J. Ophth. **2**:553, 1919.

coalesce, while concomitant atrophy develops in the area adjacent to the nerve head. It is only the final picture which might be confused with that of choroideremia, but, unlike the latter, the history is that of extremely slow progress over a great many years.

Choroidal sclerosis is a degenerative vascular disturbance of the choroid primarily, and of Bruch's membrane, the pigment epithelium and the retina secondarily. This condition is more commonly found in elderly persons with evidence of vascular changes elsewhere in the body, and the sclerotic process in the larger choroidal vessels can be seen ophthalmoscopically, especially since these are frequently set off against pigment, which has migrated into the intervascular spaces. Some of this pigment migrates into the layers of the retina as well, often lying anterior to the retinal vessels. Colloid bodies, or drusen, may appear on Bruch's membrane as an expression of degenerative changes of that structure. The disk is frequently pale and waxy, while central visual acuity, the visual field and light sense deteriorate progressively and to a marked degree.

Massive Gliosis: Verhoeff [12] implied that the diagnosis of true choroideremia depends on the complete absence of choroid except for the macular region, the absence of pigment and an absolutely white sclera, visible throughout the fundus. He claimed that many so-called cases are in fact cases of retinitis pigmentosa with choroid intact but hidden from view by widespread, deep-seated retinal gliosis. By way of illustration, he mentioned 5 cases of retinitis pigmentosa in one family, in 2 of which (both of males) the fundi closely resembled the classic conception of choroideremia, and he suggested that widespread gliosis accompanying retinitis pigmentosa is a male characteristic.

Careful study of the fundi in the present case, as well as of the paintings in the papers of Parker and Fralick and of Benjamin Friedman, leads me to doubt greatly that gliosis in these cases could have been so widespread and massive as to produce fundi of such pallor and yet fail to obscure those few choroidal vessels outside the macular region. In all three papers choroidal vessels may be seen as they perforate the sclera, testifying to their depth in the choroid. Surely, gliosis sufficient to obscure the more superficial choriocapillaris and pigment epithelium should have hidden these deeper vessels as well. I would suggest this point as a criterion for differentiating massive gliosis and choroideremia.

SUMMARY

A case of choroideremia is presented in a white man aged 29 with moderate myopia and some macular degenerative changes in one eye with

12. Verhoeff, F. H.: Retinitis Pigmentosa with Widespread Gliosis: So-Called Choroideremia, Arch. Ophth. **27**:688 (April) 1942.

resultant acuity of 20/200, while vision in the opposite eye was correctible to 20/30. The salient points of typical choroideremia are outlined, and the more recent cases in the literature (published since Bedell's review, in 1937) are briefly summarized. In the absence of histologic data, some of the published speculations regarding the cause and nature of the disease are reviewed. Finally, a brief differential diagnosis of choroideremia, retinitis pigmentosa, gyrate atrophy, choroidal sclerosis and massive gliosis is presented.

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MYOPIA INVERSA

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Translated from the German by Erich Pressburger, M.D., San Francisco

A CONUS inferior, which is spoken of in English-speaking countries as the Fuchs coloboma, after my father, is located below and either to the temporal or to the nasal side of the disk. It sometimes has a horizontal [1] or a somewhat oblique straight line at the edge of the disk and is characterized by the fact that the lower half of the fundus usually has less pigmentation than the upper half. The error of refraction of the lower half of the fundus is also frequently greater than that of the upper. Myopia is found in a majority of the cases. In histologic sections, a thinning of the sclera has been noted in the lower quadrants.[2]

A nasal conus [3] occurs less frequently than the inferior conus and often shows a lesser degree of pigmentation of the fundus on the nasal side and occasionally a bulging of the sclera, which can be determined by the difference in refraction of this area. In the cases of nasal conus the fovea frequently lies closer to the disk than normally. While the blindspot is situated in the normal position with respect to the fixation point, my father was able to observe in 2 cases on histologic examination that there was a considerably shorter distance between the margin of the disk and the fovea.[4] There is nearly always an inverse type of arrangement of vessels in the cases of nasal conus, and in these cases a medium or a great degree of myopia is apt to be present. The following cases indicate that myopia has a special connection with the nasal conus.

CASE 1.—A woman aged 51 had a large nasal conus in the right eye adjoining a round disk, without any inverse type of vessel arrangement, and a myopia of 15 D., while in the left eye there was a normal fundus with normal refraction.

CASE 2.—A man aged 54 had a nasal conus and a myopia of 10 D. in his right eye, while in the left eye he had an inferior and a nasal conus and a myopia of 7 D.

1. Fuchs, E.: Lehrbuch der Augenheilkunde, ed. 17, Vienna, F. Deuticke, 1944, illus. 10, pl. 1.

2. Fuchs, A.: Die Erkrankungen des Augenhintergrundes, Vienna, F. Deuticke, 1943, illus. 1, pl. 4.

3. Fuchs,[2] illus. 4, pl. 1.

4. Fuchs, E.: Klin. Monatsbl. f. Augenh. **62**:433, 1919.

As Roenne[5] has demonstrated, in cases of nasal conus the nasal parts of the fundus show a greater degree of myopia than the macular region or the temporal fundus.

There are occasional cases in which changes occur at the disk which resemble the nasal conus but which are something quite different. They have not attracted much attention until now, but they are entitled to special notice. In these cases, on the nasal side of the optic nerve one finds changes which look exactly like those on the temporal side of the optic nerve in cases of high myopia, namely, large atrophic zones of the choroid (circumpapillary atrophy), which extend toward the nasal side more than toward the temporal side. As such eyes are myopic, I propose to call these changes "myopia inversa." In the course of twenty years, I have made notes on 14 such cases and have drawn schematic sketches of the fundi.

CASE 1.—A woman aged 75 had vision of 6/18 in the right eye with a —4 D. lens. The disk showed an inverse type of vessel arrangement, and at the nasal margin of the disk there was a sharply defined zone, measuring 3 by 1½ papilla diameters (p. d.). This zone was white and somewhat yellowish in parts, partly dotted and more extensive in the upper than in the lower region (fig. 1). There were no changes in the vessels in this region. Below the disk there was a blurred, reddish area, ¾ p. d. in size, next to the margin of the disk. The nasal half of the fundus had a lighter pigmentation and appeared albinotic, while temporal to the disk the fundus was tessellated. Retinoscopic examination could not be performed because of heavy opacities in the vitreous. The fovea seemed to lie closer to the margin of the disk than normally. The blindspot was enlarged at the top and even more so at the bottom. There was no change in its position, however. The left eye showed a myopia of 1.5 D. and had a wider zone of atrophy of the pigment epithelium at the nasal side of the disk, but not so broad a one as the zone in the right eye. An inverse type of vessel arrangement was also present. In both eyes this disk looked perfectly round.

CASE 2.—A 14 year old boy when seen in December 1925 had a refraction of —3.5 D. sph. ⌒ —1.25 D. cyl., axis 20 in the right eye. The disk showed an inverse type of vessel arrangement and was somewhat oval. At the nasal margin of the disk there was a half-moon-shaped area of atrophy of the pigment epithelium. The pigmentation of the fundus was not as pronounced nasally as temporally. The fovea seemed to be normally distant from the disk. In the left eye, there was a refraction of —3.00 D. sph. ⌒ —2.5 D. cyl., axis 10. The left disk showed only an inverse type of vessel arrangement and had a long, vertical oval form; but here again the nasal fundus was less pigmented than the temporal. In 1943 I was able to examine this patient again, after an operation had been performed elsewhere for divergent strabismus. The operation was not entirely successful, as he still had a divergence of 20 degrees. The patient at this time was 32 years of age. The refraction in his right eye was —8.00 D. sph. ⌒ —1.00 D. cyl., axis 15. Thirty degrees nasal to the fovea, the refraction was —15.00 D. sph. ⌒ —1.00 D. cyl., axis 25. The atrophic zone had become larger and surrounded the disk. Nasally, this zone was 1½ p. d. wide, while on the temporal

5. Roenne: Klin. Monatsbl. f. Augenh. **57**:512, 1916.

side it was extremely narrow (fig. 2). Above the fovea, a small, white atrophic spot had developed. A similar white spot lay nasal to the disk and had an irregular pigmentation, like a small pigment dot, which also showed up in the yellowish atrophic zone. The left eye had a refraction of —2.5 D. sph. ⌒ —2.00 D. cyl., axis 15. Thirty degrees nasally from this fovea, the refraction was —10.00 D. sph. ⌒ —3.00 D. cyl., axis 177. The disk showed the same vertical oval formation, with an inverse type of vessel arrangement, as previously mentioned. In both eyes, the lack of pigment in the nasal fundus was very conspicuous. The fovea seemed to be at a normal distance from the disk. This was the only case in which I had an opportunity to make examination after a long interval. I noted a definite increase of the myopia in the right eye, with a nasal atrophic zone, and this had become appreciably larger. When I saw the zone on the patient's fourteenth birthday, I noted on the nasal side only a white, half-moon-shaped area, but in his thirty-second year circumpapillary atrophy had developed. In addition, a pigment spot had formed in the atrophic section, and other atrophic spots could be discerned in the fundus. From the fact that this eye alone disclosed a considerable increase of myopia, while the other, with a negligible increase in the refraction, showed no change in the disk, one might deduce that there was a real connection between the atrophic zone and the myopia; evidently, the atrophic zone was already considerably developed at a time when the myopia of the posterior pole was insignificant. Two points were interesting. First, the patient had a divergent squint of the left eye; this eye had no particular myopia and had become amblyopic. His vision of 6/8 in 1925 dropped to perception of hand movements without any changes in the fovea. One would have expected just the opposite, namely, that he would be amblyopic and would squint with the right eye, this eye being myopic and showing pronounced changes in the fundus. Second, in spite of the marked difference in the refraction of the posterior pole, there existed a considerable degree of myopia of the nasal fundus, as a result of the bulging of the nasal sclera in both eyes. On retinoscopic examination, it was obvious that in the left eye a much higher degree of astigmatism appeared in abduction than when the patient looked straight ahead. In the right eye I found the same astigmatism in the two positions, namely, in abduction and when the patient looked straight ahead. In the left eye, however, not only did I find an increase of from 2 to 3 D. in cylinder, which might have been due to marginal astigmatism, but I also observed that the axis was 15 degrees and the axis of the nasal fundus was 177 degrees. This striking discrepancy of the axes was probably due to the torsion of the eye when turned to the side.

Case 3.—A man aged 56 had a refraction of —2.25 D. sph. ⌒ —1.00 D. cyl., axis 180 in the right eye, an inverse type of vessel arrangement, an oval and oblique disk and a large conus on the temporal side and below. He also had arteriosclerotic retinopathy with occasional white spots and hemorrhages (fig. 3). In the left eye I found his refraction to be —3.00 D. sph. ⌒ —2.50 D. cyl., axis 10; here, too, the disk was oval but in a vertical position. The distribution of the vessels was inverse, and nasally there was an adjacent atrophic zone about 2 p. d. wide and 2¼ p. d. high. At the same time, three fourths of the disk was surrounded by the atrophic zone. Several retinitic spots were also found, with no marked difference in the pigmentation between the nasal and the temporal fundus.

Case 4.—A woman aged 63 complained that objects were distorted with her right eye. Vision in the right eye was 6/60 with a correction of —4.00 D. sph. ⌒ —1.00 D. cyl., axis 180 and 6/60 in the left eye with a —4.00 D. lens. In both

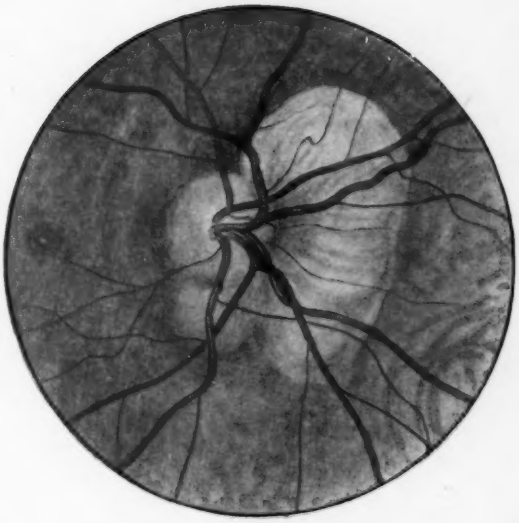

Fig. 1.—Fundus in case 1.

UNIV. OF MICH.

eyes there was an absolute central scotoma, and the right eye showed metamorphopsia. Both disks were round; the distribution of the vessels was inverse, and at the nasal side of the disk there was a cleancut, yellowish region, 2½ p. d. wide, where there had evidently been absorption of pigment epithelium. In the macula of the right eye, there was a small Fuchs spot. In the left eye, a some-

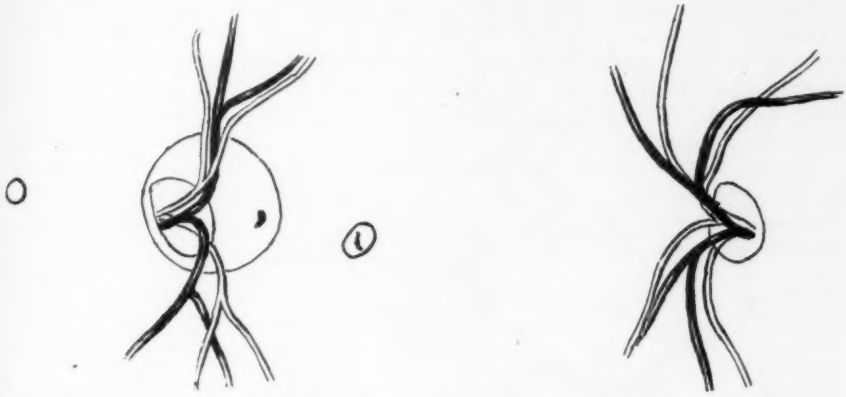

Fig. 2.—Right eye: — 8.00 D. sph. ⌒ — 1.00 D. cyl., axis 15; left eye — 2.50 D. sph. ⌒ — 2.00 D. cyl., axis 15.

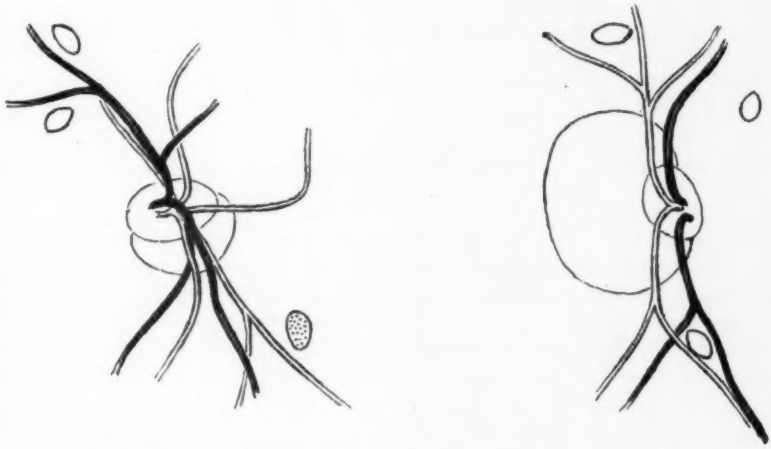

Fig. 3.—Right eye: — 2.25 D. sph. ⌒ — 1.00 D. cyl., axis 180; left eye: — 3.00 D. sph. ⌒ — 2.50 D. cyl., axis 10.

what larger, white, cleancut spot was visible in the macula, possibly an old Fuchs spot. An incipient cataract made retinoscopic examination impossible (fig. 4).

CASE 5.—A middle-aged woman had a correction of — 7.00 D. for each eye, giving vision of 6/18? Each eye had a slightly vertical, oval disk, with an inverse arrangement of the vessels; the disk was surrounded by a clear zone, resembling circumpapillary atrophy on the temporal side, where it was rather

narrow. On the nasal side, the zone measured 1½ p. d. Figure 5 *A* shows the left eye. One can see a considerable conus of supertraction and a blur of the temporal margin of the disk, as a result of which the atrophic zone appears wider than it really is. In the nasal part of the atrophic zone a small retinal artery originates, which, although it does not show a hook or bow at its source, most likely springs from the choroid. Nasally the fundus in both eyes was albinotic, strikingly resembling the fundus in a myopic eye and showing in the left eye an atrophic posterior vortex vein. In the left eye, 3 p. d. nasally and below the disk the fundus was very bright; temporally the fundus was reddish brown. Retinoscopic examination of the left eye showed a refraction of —4.50 D. sph. ⌒ —5.50 D. cyl., axis 162. Twenty degrees nasally from the disk the refraction was the same. The fovea seemed to be located closer to the temporal margin of the disk than normally. The blindspot revealed an enlargement in the right eye in all directions. The blindspot of the left eye extended from 9 to 21 degrees (fig. 5 *B*). Whereas in all cases previously mentioned an atrophic zone was found chiefly on the nasal side, in the last case a very pronounced circumpapillary zone of

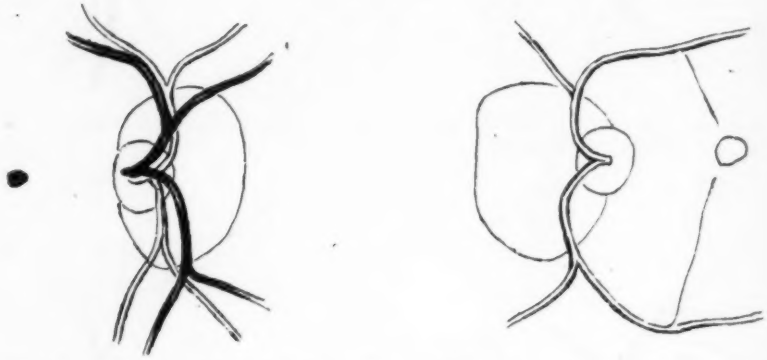

Fig. 4.—Right eye: —4.00 D. sph. ⌒ —1.00 D. cyl., axis 180; left eye: —4.00 D. sph.

atrophy was present in both eyes, which was greatly extended toward the nasal side. An area on the nasal side was distinctly albinotic in type. Retinoscopic examination in the horizontal meridian revealed the same refraction nasal to the disk and at the posterior pole; but, as the fundus was especially light nasally and below, one could have been confronted with a high myopia. It seems that in this instance the blindspot extended quite a bit closer to the fixation point than normally.

Case 6.—A middle-aged woman had 5/5 vision in the right eye with —0.5.00 D. cyl., axis 100. She had an inverse type of arrangement of the vessels, a round disk and a small nasal conus. Vision of the left eye was 5/12 with a correction of —5.00 D. sph. ⌒ —1.50 D. cyl., axis 140. The vessels showed an inverse arrangement, and the disk was vertically oval, looked somewhat smaller than normal and was decidedly dark red. It was surrounded by an atrophic zone, rather narrow on the temporal side but nasally over 1 p. d. in width (fig. 6).

Case 7.—A middle-aged woman had a myopia of —5 D. in each eye and there was a large nasal atrophic zone, which in the right eye broadened somewhat on top and in the left eye spread out nasally and horizontally. In the right eye a cilioretinal vessel originated at the temporal margin of the disk and extended toward

the macula, while at the lower margin of the disk in the left eye a small, hook-shaped vessel, similar to a cilioretinal vessel, could be seen running downward. Pigmentation was the same on the two sides. The distribution of the vessels was inverse (fig. 7).

CASE 8.—A man aged 54 had scars on both corneas, which made retinoscopic examination impossible. He had 5/18 vision in the right eye with a — 10.00 D. lens

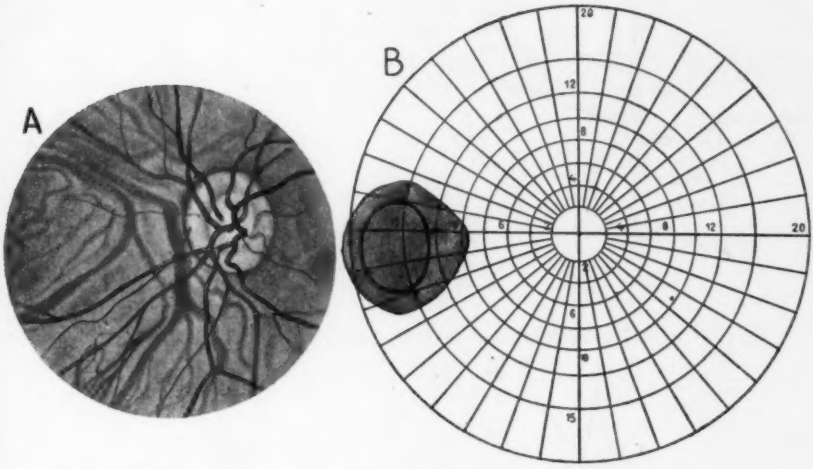

Fig. 5.—*A*, fundus of the left eye; *B*, field of the left eye, with enlargement of the blindspot.

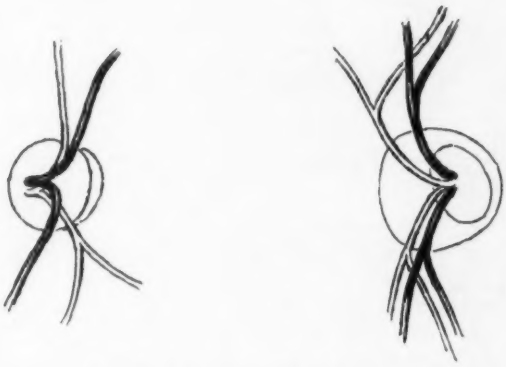

Fig. 6.—Right eye: — 0.50 D. cyl., axis 100; left eye: — 5.00 D. sph. ⌒ — 1.50 D. cyl., axis 140.

and nasal to the disk there was an atrophic zone 1 p. d., wide (fig. 8). This eye was extremely divergent. Vision in the left eye was 5/18 with a — 7 D. lens, and an atrophic zone extended nasally downward from the oblique, oval disk, ending in a pinpoint. The outline of this zone was rather blurred above. The distribution of the vessels was inverse on both sides, and the pigmentation of the fundus was the same throughout.

Case 9.—A man aged 69 had 5/12 vision in the right eye with a — 10.00 D. ⌒ 1.00 D. cyl., axis 180. There were a normal distribution of the vessels and circumpapillary atrophy, which was more marked on the nasal side than on the temporal. The pigmentation of the fundus was the same in the two eyes. With — 1.00 D. sph. ⌒ — 1.50 D. cyl., axis 100, vision in the left eye was 5/6. There was an atrophic zone 1½ p. d. wide at the nasal side. At the upper margin of

Fig. 7.—Right eye: — 5.00 D. sph.; left eye: — 5.00 D. sph.

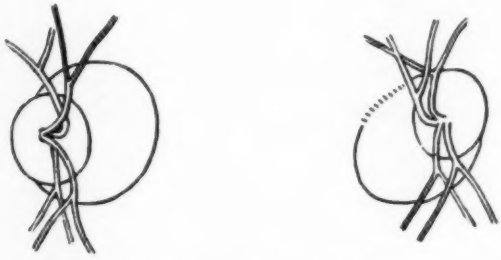

Fig. 8.—Right eye: — 10.00 D. sph.; left eye: — 7.00 D. sph.

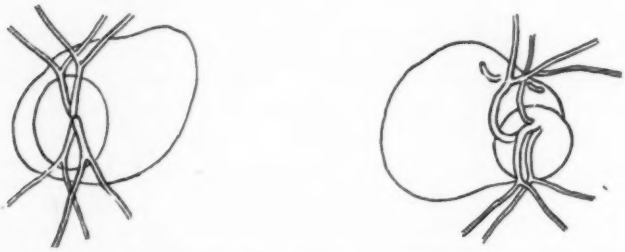

Fig. 9.—Right eye: — 10.00 D. sph. ⌒ — 1.00 D. cyl., axis 180; left eye: — 1.00 D. sph. ⌒ — 1.50 D. cyl., axis 100.

this zone heavy choroidal vessels could be seen. The distribution of the retinal vessels was inverse (fig. 9). Retinoscopic examination of the left eye gave the following refraction: fovea, — 1.00 D. sph. ⌒ — 2.00 D. cyl., axis 100; nasal to the fovea, — 3.00 D. sph. ⌒ — 3.00 D. cyl., axis 100; temporal to the fovea, + 0.50 D. sph. ⌒ — 2.50 D. cyl., axis 115. There existed a slight ectasia of the nasal sclera.

CASE 10.—A man aged 31 had 5/8 vision in the right eye with a correction of — 11 D. There was an atrophic zone of the choroid 1½ p. d. wide on the nasal side. The nasal half of the fundus of each eye was considerably less pigmented than the temporal half. There was an inverse arrangement of vessels in both eyes. Vision in the left eye was 5/8? with a — 10.00 D. lens. At the nasal side of the disk there was an atrophic zone ¾ p. d. wide (fig. 10). Retinoscopic examination revealed the following refraction: Right eye: fovea, — 10.00 D.; 30 degrees temporally, — 6.00 D.; 30 degrees nasally, — 14.00 D. Left eye: fovea, — 10.00 D.; 30 degrees temporally, — 6.00 D.; 30 degrees nasally, — 15.00 D. There was, therefore, a pronounced ectasia of the nasal scleral region, which was somewhat greater in the eye with the large atrophic zone. The difference in the refraction temporally and nasally was 6 to 8 D., and was consequently considerable.

Fig. 10.—Right eye: — 11.00 D. sph.; left eye: — 10.00 D. sph.

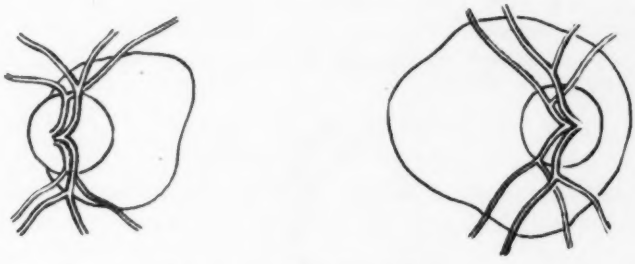

Fig. 11.—Right eye: — 5.00 D.; left eye: — 4.50 D.

CASE 11.—A man aged 73 had tiny corneal scars on both eyes. Vision in the right eye was 5/12 with a — 5.00 D. lens, and nasal to the disk he had an atrophic zone 1 p. d. in width. In the left eye vision was 5/12 with a — 4.50 D. lens (fig. 11), and an area of circumpapillary atrophy developed from the nasal atrophic zone, which was ¼ p. d. wide temporally and 2 p. d. wide nasally. An inverse arrangement of vessels was present in both eyes. The fundus showed only a little difference in the pigmentation between the nasal and the temporal half.

CASE 12.—A man aged 42, whose vision was 5/5 in the right eye with — 5.50 D. sph. ⌒ — 1.00 D. cyl., axis 50, had a reddish, half-moon-shaped halo nasal to the disk, measuring about 1 p. d. and bordered by a narrow, whitish band. He had an inverse arrangement of vessels, and the pigmentation of the fundus was the same throughout. In the left eye, vision was 5/5 with a — 5.5 D. sph. ⌒ — 1.00 D. cyl., axis 180, and there was the usual temporal conus (fig. 12).

CASE 13.—A woman aged 30, with a refraction of — 10.50 D. sph. ⌒ — 1.00 D. cyl., axis 15, in her right eye, had vision of 5/12. There was an atrophic zone, 1 p. d. wide at the nasal side of the disk, which nearly surrounded the disk. In the left eye, vision was 5/12 with a correction of — 9.50 D. sph. ⌒ — 1.00 D. cyl., axis 20, and there was a similar atrophic zone, 1 p. d. wide nasal to the disk, which was wider in the upper than in the lower half. Both eyes had an inverse type of vascular arrangement, and the fundi were albinotic on the nasal side and reddish brown on the temporal side, so that the choroidal vessels were not visible here (fig. 13).

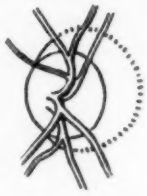

Fig. 12.—Right eye: — 5.50 D. sph. ⌒ — 1.00 D. cyl.; axis 50.

Fig. 13.—Right eye: 0.50 D. sph. ⌒ — 1.00 D. cyl., axis 15; left eye: — 9.50 D. sph. ⌒ — 1.00 D. cyl., axis 20.

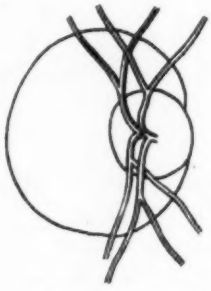

Fig. 14.—Left eye: — 11.00 D. sph.

CASE 14.—A woman aged 70 had a mature cataract in the right eye, and in the left eye vision was 5/18 with a correction of — 11.00 D. In addition to an incipient cataract and many opacities in the vitreous of the left eye, there was an atrophic zone in the choroid running nasally from the disk, 1½ p. d. wide, and almost completely surrounding the disk. The vessels were inverse in distribution, and the pigmentation of the fundus was the same throughout (fig. 14).

The cases described here are not all that I have seen. They occurred so often that I ceased to make a record of all of them. When I started

to collect data on them in 1926, I made notes of 6 instances, of which only two sketches and a colored picture are available. In 1944, I have memoranda on 4 cases (9 to 13). In all these cases I observed an extended light, sharply defined area at the nasal side of the disk, corresponding to what one finds at the temporal side in high myopia. I therefore call this type of change in the fundus "myopia inversa." One is dealing here with a zone in which the pigment epithelium has become atrophic, so that the choroid and sclera shine through with a yellowish or whitish color, and in which the choroid seems to be everywhere atrophic (fig. 1). Usually this atrophic zone lies nasal to the disk in a straight line, and only occasionally runs slightly oblique, either nasally and above or nasally and below (figs. 7 and 9), but joins the disk on the nasal side. Thus, this zone, with its exact nasal position, is distinguishable from the congenital conus on the nasal side, since the latter rarely lies strictly on the nasal side, but more often is nasal and below. The outline of the nasal zone is usually clear everywhere (fig. 12). Only once did it appear intermittently blurred (fig. 8). The atrophic zone in myopia inversa generally is considerably larger than one would expect from the amount of refraction. I have never found a small, half-moon-shaped atrophic zone on the nasal side of such a degree as that which one ordinarily sees with myopia of 3 to 4 D., and which is perhaps ¼ to ⅓ p. d. wide. Often, the nasal zone is rather wide, 1 to 2 p. d., and not infrequently the atrophic zone surrounds the disk, analogous to circumpapillary atrophy, as I had an opportunity to observe in 5 cases (figs. 2, 5, 6, 9 and 11).

The distribution of the vessels is nearly always inverted. Once (fig. 9) I found a normal distribution of vessels in the right eye, which showed circumpapillary atrophy with a marked nasal extension. The myopia of that eye was —10 D., while examination of the left eye disclosed a myopia of —1.00 D. ⌒ —1.50 D. cyl., axis 100, with a nasal atrophic zone 1½ p. d. wide and an inverse arrangement of vessels. The circumpapillary zone on the nasal side with a normal distribution of the vessels of the right eye in a case of high myopia leads me to presume that such an instance represents a transition from ordinary myopia to myopia inversa.

Pronounced supertraction of the temporal margin of the disk is not present ordinarily in cases of myopia inversa. In case 5 only was one able to see a certain supertraction of the temporal margin of the disk (fig. 5), because this was very hazy and partially covered the vessel funnel. I found pronounced supertraction of the temporal margin in the case shown in figure 15, to which I shall refer later; in this case I found a wide nasal atrophic zone, but no myopia. Also, in a case to be mentioned later, one of inferior myopia, the upper margin of the disk was blurred and showed actual supertraction over the disk. Bor-

ders of the nasal zone are generally sharply defined only in one eye (fig. 2), the zone appearing reddish and being circled by a white band. In another case (fig. 8) part of the zone was not clearly outlined.

The nasal zone is usually homogeneous in color. I once found pigment in this region (fig. 2), and at another time choroidal vessels appeared (fig. 9); again, a vessel originated in the margin of the zone below, which formed a hook and ran down and temporally (fig. 7). From its appearance, one can be sure that one is dealing with a condition analogous to a myopic conus and circumpapillary atrophy on the temporal side, and not a congenital anomaly, in these cases in which there is an atrophic zone nasally. Case 2 furnishes convincing proof; in this case a circumpapillary atrophy developed in the patient's thirty-second year from the wide nasal zone which I had previously sketched when he was 14 years old. There was, therefore, a progressive change in the fundus.

It is manifest that a genuine nasal conus does not exist in these cases. I have never seen a combination of a typical nasal conus and an atrophic zone. In high myopia there is usually no difference between the temporal myopic conus and an extended temporal or circumpapillary atrophy of the choroid, but there is a great difference between the myopic conus and the nasal conus. A myopic conus develops slowly and becomes wider, usually about the tenth year, while the nasal conus represents a congenital abnormality and usually does not undergo any of the changes similar to those in the inferior conus. The disk was usually vertically oval in cases of myopia inversa (figs. 2 and 3) or obliquely oval (case 7), but a white crescent was never visible beside the disk, as one finds it sharply defined at the nasal margin. I seldom saw an atrophic zone below like those on the nasal side, which is remarkable, since the inferior conus can be found more frequently than the nasal conus. I shall refer to this question later. In the right eye in case 3, the wide conus seen below and inside is a congenital one and can be easily distinguished from the wide atrophic zone on the nasal side.

The fovea appeared to be closer to the disk than it is normally, but examination of the blindspot showed it to be normally distant from the fixation point but a trifle enlarged downward (case 1). As in the case of a real nasal conus, I believe that the fovea is often closer to the margin of the disk than normally. This is generally not true in the case of the nasal atrophic zone. Only in case 5 did the fovea seem to be closer to the temporal margin of the disk, and the enlargement of the blindspot extended from 9 to 21 degrees. The center of the blindspot corresponded, therefore, to the center of its real position.

In 5 cases the pigmentation of the fundus was less pronounced on the nasal than on the temporal side, while in the other cases it was the same everywhere. There is, therefore, a pigmentation of the fundus in

these cases similar to that in cases of inferior conus. In case 1 only a minor difference in the pigmentation of the two halves of the fundus existed. In case 5 a considerable difference appeared.

In these cases of myopia inversa the visual acuity varied from 6/6 to 6/60. In a few cases it was better than one usually expects with ordinary myopia of the same degree. In case 10 vision was 5/8, and in case 9 it was 5/12 with a correction of —10 D. sph. ⌒ —1 D. cyl., axis 180. The patient was 69 years old, and for a person with myopia of this age such an acuity can be considered very good.

The degree of myopia of the nasal atrophic zone is quite different. What appears conspicuous and important is that the extension of the nasal zone is caused less by the degree of myopia than is the extension of the temporal zone in ordinary myopia. In the latter, one can estimate the approximate amount of myopia from the appearance of the fundus. This cannot be judged, however, from the nasal zone, because with very slight myopia one finds large changes at the disk. In case 4 there was a particularly wide zone with a myopia of 4 D.; in case 14, a very large, comprehensive nasal zone with a myopia of 1 D., and in case 3, a zone 2 p. d. in width with a myopia of 2½ D. It is interesting to speculate on the width of the atrophic zone in relation to the amount of myopia, which can be done in case 9. In the right eye, a zone of circumpapillary atrophy existed with a myopia of —10 D., which was wider nasally than temporally. The arrangement of the vessels was normal. The left eye showed a wide nasal atrophic zone of 1½ p. d. with inverse type of distribution of the vessels and a myopia of 1 D. One could assume from the width of the atrophic zone that the myopia of the left eye should be at least as high as that of the right eye, and yet it was actually much lower.

One of the clinical characteristics of inverse myopia is an extended atrophic zone at the nasal side of the disk, which has the appearance usual with high myopia. In the course of time this zone may become much larger. Pigment spots and occasional isolated atrophic areas may appear in the fundus. At the same time, the arrangement of the vessels is inverse.

It is strange that this change at the nasal side of the disk associated with myopia has not been described in greater detail. It is not mentioned in the "Kurzes Handbuch der Ophthalmologie." My father mentioned only 1 case which he had observed, and in which there was a nasal half-moon of 2½ p. d.[6] He expressed the belief that if the disk had been surrounded and accompanied with atrophic spots it would have corresponded to the ophthalmoscopic picture of the fundus in a case of ordinary high myopia. I cannot say whether these atrophic zones develop as an ordinary circumpapillary atrophy in cases of high myopia,

6. Fuchs, E.: Arch. f. Ophth. **93**:381, 1917.

in which a temporal conus first becomes larger and wider and finally encloses the disk, but it is very probable. In case 2, however, in which the patient was observed when he was 14 years old, a very large area of atrophy lay at the nasal side of the disk. In cases of high myopia at this age, such zones can hardly be so well developed. It is remarkable that a difference in refraction exists in the various parts of the fundus, which may very often be found in cases of myopia inversa. In 4 cases (2, 5, 9 and 10) I determined the refraction of the posterior pole of the eye by retinoscopy and that at a distance of 30 degrees nasally therefrom. The refraction in the right eye in case 2, in which a wide and nasal circumpapillary atrophy existed, was 7 D. greater nasally than in the posterior pole of the eye. The left eye had also a myopia of approximately 8 D. higher on the nasal side, but had no nasal zone, although an inverse arrangement of vessels was present. There is here a peculiar contradiction. The amounts of refraction of the various parts of the fundus are practically the same, whereas the picture of the fundus and the appearance of the disk are entirely different. The right eye had become much more myopic from the fourteenth to the thirty-second year in the case of this patient. The refraction increased from —3.50 to —8.00 D, while the slight myopia of the left eye remained practically unchanged. Therefore, a progressive myopia existed in the right eye, while the myopia in the left eye remained stationary. One could assume that a certain congenital myopia existed in both eyes, together with differences in refraction in the fundus and an inverse type of vessel arrangement, and that the circumpapillary atrophy of the right eye was attributable to the progressive myopia.

In case 9, the difference in refraction between the temporal and the nasal side was 4½ D. In case 10, the difference between the temporal and the nasal side was 8 D. in the right eye and 9 D. in the left eye, and the difference between the fovea and the nasal side was 4 D. in the right eye and 5 D. in the left eye. One can see from these findings that there exists an ectasia of the nasal part of the sclera, where the macular regions sometime participate. At other times, the nasal side shows no higher refraction than the temporal parts of the fundus. One might believe that the atrophic zone on the nasal side was caused by this ectasia, but this does not always seem to be the case, for in another instance the refraction at the posterior pole and that of the nasal part of the fundus were equal. In this case, a narrower zone of circumpapillary atrophy existed than in the former case, and the nasal ectasia was not so extensive. The nasal bulge in the sclera does not seem to be the only cause of the atrophic zone on the nasal side of the disk. This is supported by the fact that considerable differences of refraction existed in those eyes in which no conus of the disk was revealed. Rubert[7] described

7. Rubert: Ztschr. f. Augenh. **20**:512, 1916.

2 such cases in which the disk was normal, except for an irregular type of vessel arrangement, and in which the refraction was considerably higher below than above. In 1 case, refracton showed slight hyperopia, while below the myopia increased to —8.00 D. In the other eye, a slight myopia was found above, while below there was a pronounced myopia. Such cases have rarely been observed, although they probably occur more frequently than one might suppose.

The following case[8] falls under the head of myopia inversa. A woman aged 32 had had poor vision from childhood and at 14 years of age wore her first pair of glasses, which were only slightly increased later. Her parents and grandparents had not been myopic. The disk and the distribution of the vessels were normal in both eyes. The fundus was reddish brown, but nasally it suddenly became conspicuously tessellated, most likely due to a small amount of pigment in the pigment epithelium. Vision in the right eye was 5/8 with a —11.00 D. lens; in the left eye it was 5/8 with a —6.00 D. lens. The cornea of the right eye had a refractive power of 60.5 D., and that of the left eye, 62 D. in the horizontal meridian. In the right eye, retinoscopic examination disclosed a refraction of —11.00 D. ⌒ —1.50 D. cyl., axis 25, and in adduction of 30 degrees, a refraction of — 14.00 D. ⌒ 0.75 D. cyl., axis 25. In the left eye, retinoscopic examination disclosed refraction of —6.50 D. ⌒ —1.00 D. cyl., axis 180 at the posterior pole, and in adduction of 30 degrees, a refraction of —9.00 D. ⌒ 0.50 D. cyl., axis 180. It is apparent that in this case there was a special, congenital myopia, which increased negligibly, which was common but in which the nasal part of the sclera showed a remarkable bulge. There existed neither any abnormality of the disk nor any myopic stretching of the choroid. In myopia inversa, the size of the nasal zone is also remarkable. Why is it so large, even with minor degrees of myopia? The nasal ectasia may cause a special pull of the choroid, because here, between the nasal part of the ora serrata and the disk, there is a much shorter section of the choroid, and so the lamina vitrea and pigment epithelium are subject to more pull than in ordinary myopia, in which the pulling is effective on the temporal side and thus the entire posterior portion of the eyeball is included. As previously stated, in 1 case (case 9) I found no particular bulge on the nasal side on retinoscopic examination; therefore, there must be another important reason for the special pulling on the nasal side. Probably in cases of myopia inversa the optic nerve passes through the sclera in a reverse direction, for there always exists an inverse type of arrangement of the vessels in these cases. Perhaps this exposes the nasal half of the disk more to the pulling.

I must mention a case here (Negrusch) in which the clinical picture approximated that of myopia inversa; only, the myopia was lacking. This

8. Fuchs,[2] p. 169.

would contradict the view that a slight extension of the axis with a reversed direction of entrance of the optic nerve has any connection with the origin of the nasal zone. A woman aged 67 had vision of 5/8 with —1.50 D. cyl., axis 150 in the right eye and of 5/8 with —0.50 D ⌣ —1.75 D. cyl., axis 140 in the left eye. She had marked supertraction of the temporal margin of the disk in both eyes and a very wide and high atrophic zone at the nasal side of the disk in the left eye. The arrangement of the vessels was inverse in both eyes. The pigmentation was the same everywhere, and the fovea was normally distant from the margin of the disk (fig. 15). Because of an external disease of the eye, retinoscopic examination could not be performed. The eye presented a perfect picture of myopia inversa; perhaps there was a moderate lengthening of the axis, but this was compensated for by an especially flat lens or by a moderate curvature of the cornea.

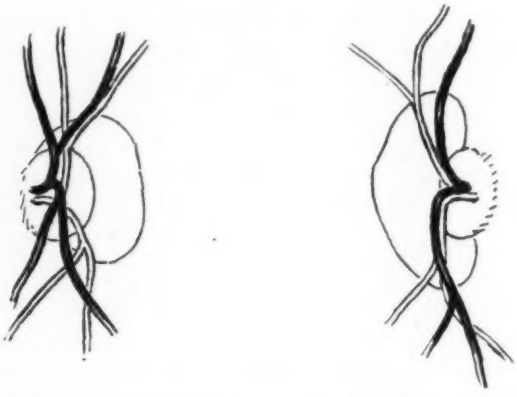

Fig. 15.—Right eye: —1.50 D. cyl., axis 150; left eye: —0.50 D. sph. ⌣ 1.75 D. cyl., axis 140.

In this connection, I wish to draw attention to these cases in which a similar atrophic zone can be found beneath the disk with myopia. They seem to be rare, and I have a record of only 2.

Case 1.—A man aged 39 had a small, typical inferior conus in his right eye, while in his left eye he had a wide, half-moon-shaped atrophic zone, 1½ p. d. in width downward and a myopia of 4 D.[8]

Case 2.—A woman aged 56 consulted me because of diplopia. Examination revealed an incipient cataract in each eye, which had produced a monocular diplopia. The right eye had vision of 5/12 with a —4.50 D. lens and an inverse arrangement of the vessels; the disk was slightly oblique and oval, and there was a wide atrophic zone of the choroid with a small pigment spot downward, measuring about 1½ p. d. in width (fig. 16). In the left eye vision was 5/12 with a —4.50 D. lens, but here the distribution of the vessels was normal. Below the disk, a half-moon-shaped atrophic choroidal zone, ¾ p. d. in width, could be seen with

a longitudinal pigment spot at its margin. In both eyes, the margin of the disk which lay opposite the atrophic zone was blurred and looked like an area of supertraction. There was no difference in the pigmentation of the various parts of the fundus. Retinoscopic examination was impossible because of irregularities of the lens.

It seems to me that this feature, which I might call myopia inferior, is rarer than myopia inversa, a fact which is astonishing, since the real, congenital, inferior conus is so much more frequent than the real, congenital, nasal conus. It would seem that these atrophic zones found at unusual places do not originate from a congenital conus; neither are they the offspring of a genuine conus; nor do they have any connection whatever with them, but they are due to a pull, and this pulling seems to be more frequent nasally than below.

As to the histology of the nasal conus, I found two publications in which, unfortunately, no ophthalmoscopic examination had been done. One record is my father's,[6] and the other is Salzmann's.[9] The findings

Fig. 16.—Right eye: —4.50 D.; left eye: —4.50 D.

are very different. In the case of Salzmann the ophthalmoscopic picture showed a temporal conus, in which the optic nerve entered obliquely into the retina. The nasal margin of the porus opticus formed a wide angle, while there was a marked supertraction at the temporal margin, where the lamina vitrea and the limiting tissue far overreached the walls of the porus opticus. In the picture, one has the impression that the optic nerve in the porus opticus makes a sort of bend, in which the nerve fiber bundles approach nasally and turn just in front of the lamina toward the nasal side, just behind the cribriform plate. Histologic examination by my father revealed no enlargement of the porus opticus on the nasal side, but showed a conical narrowing. The retina was somewhat drawn over the porus opticus toward the nasal side, the lamina vitrea stopping at the limiting tissue and the ganglion cell layer and the nuclear layer being drawn over the nerve tissue of the disk. At the nasal side, the glass membrane began at the scleral opening, but

9. Salzmann, M.: Arch. f. Ophth. **143**:568, 1941.

the pigment epithelium and the external layers of the retina were missing for quite a distance. Thus, one has two different kinds of nasal conus, as well as two similar forms of temporal conus: first, the broadening of the porus opticus where the broadened wall of the canal can be viewed from the front as a white half moon and, second, an atrophy of the pigment epithelium, by reason of which the choroid can be seen as a lighter, rather yellowish half-moon. The latter can also be seen in cases of senile conus at the temporal side. I have seen both forms in the inferior conus.

Salzmann expressed the belief that the nasal conus often is narrow and its outlines are blurred because the nerve fiber layer which passes the nasal margin of the optic foramen is considerably thicker on the nasal side than on the temporal side. The nasal conus is often very narrow and blurred, possibly because the broadening of the porus opticus is only slight. This is not true in cases with an atrophic zone of pigment epithelium, as the illustrations show. In these cases the borders are very sharp. I am not sure whether the nerve fiber layer in a nasal conus is really thicker at the nasal than at the temporal side. This cannot be deduced from figure 6 of Salzmann's article. Perhaps here is a condition similar to that in an inferior conus, in which the retina is thicker at the upper margin of the disk than at the lower.[2] It has been assumed that the inferior conus has a relation to torsion of the eye, specifically, to the entrance of the optic nerve. It is certain that in an inferior conus the lower half of the disk discloses early pathologic changes in certain diseases just as pronouncedly as the temporal half does in a normally formed disk. I have found this in cases of glaucoma and of atrophy of the optic nerve. In the early stages of glaucoma with an inferior conus, the first depression of the cupping is discovered below, and not temporally.[10] If, in the case of disseminated sclerosis, pallor of the disk sets in and an inferior conus is present, one finds the pallor not in the temporal half, as is usually the case, but below. It seems that in an inferior conus the nerve fiber layer is thicker at the upper margin of the disk than below, for I have seen a Weiss-Otto reflex above and not nasally, where it is usually found.[11] This reflex is explained by the thick nerve fiber layer, which produces a prominence at the margin of the disk, and one has to assume that if this reflex is found to be displaced the thickest part of the nerve fiber layer corresponds to it.

It would be advisable to study the distribution of the nerve fiber layer bundles in a nasal conus in the red-free light in order to get a proper idea regarding their distribution and course.

10. Fuchs,[2] illus. 9, pl. 2.

11. Fuchs,[2] illus. 1, pl. 5.

A myopia inversa, with the extended atrophic zone at the nasal side and the inverse type of vessel arrangement, leads one to assume that the distribution of the nerve fiber bundles is different and their course more twisted than is normally the case. The extended nasal atrophic zone in myopia inversa seems therefore to correspond with the observation of my father, namely, that there is a simple atrophy of the pigment epithelium. With regard to the histology of myopia inferior, I have found a record of this rare occurrence by Salzmann. He described the case of a myopia with an inferior conus and an atypical staphyloma in the same direction.[12]

12. Salzmann, M.: Arch. f. Ophth. **54**:337, 1902. The original text was not available to me.

A NEGLECTED CAUSE OF SECONDARY GLAUCOMA IN EYES IN WHICH THE LENS IS ABSENT OR SUBLUXATED

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GLAUCOMA developing in an aphakic eye, either spontaneously or following discission of a membrane, may be due to one of many factors. Among those most frequently mentioned are delayed reformation of the anterior chamber, prolapse of the iris, incarceration of the lens capsule or iris in the wound, adhesion of hyaloid membrane to the wound and anterior synechia of an iris pillar, each resulting ultimately in extensive peripheral anterior synechias. Increased formation of aqueous from irritation of the ciliary body, such as that resulting from traction during the needling operation, has been postulated. Retained lens cortex or vitreous in the anterior chamber may mechanically block the angle and cause hypertension. The glaucoma may sometimes be secondary to iritis or uveitis.

When the lens is subluxated, it may press the iris forward on one side, so as to block the angle here. When the lens is completely dislocated posteriorly, the glaucoma which sometimes occurs has been considered to be due in some cases to increased formation of aqueous from a ciliary body mechanically irritated by the free-floating lens.

All ophthalmologists are familiar with the fact that when the lens is in situ seclusion of the pupil, with or without iris bombé, often causes secondary glaucoma. It is common experience that this form of secondary glaucoma can be relieved if communication is reestablished between the chambers by transfixion of the iris or by iridectomy, unless the condition has persisted so long that the angle is permanently closed. It is perhaps not so generally recognized that a similar cause for glaucoma can exist in eyes in which the lens is absent or dislocated. It is the purpose of this communication to discuss this type of secondary glaucoma and to present illustrative cases from the literature and from our own experience. Four types can be observed. Though all four types are very similar, the mechanism of the block is slightly different in each, and they can be more easily considered separately.

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1. Aphakic eyes, with or without iridectomy, in which the pupil is blocked by a membrane partly or wholly inflammatory.

2. Aphakic eyes, nearly always without iridectomy, in which the pupil is blocked by a herniation of vitreous through it, usually after discission of secondary cataract.

3. Eyes in which after intracapsular extraction of the lens the pupil is blocked by adhesion of the iris to the intact hyaloid membrane.

4. Eyes with partial or complete subluxation of the lens backward, in which the pupil is blocked by a herniation of vitreous through it.

TYPES OF SECONDARY GLAUCOMA ASSOCIATED WITH ABSENCE OR SUBLUXATION OF THE LENS

TYPE 1.—*Aphakic eyes, with or without iridectomy, in which the pupil is blocked by a membrane partly or wholly inflammatory.*

The first to recognize this type of glaucoma was Bowman,[1] in 1865. He stated:

> But probably the iris will be found to be more or less bulged forward, showing accumulation of fluid behind it in a space not communicating through the pupil with the anterior chamber. Often this bulging is partial or unequal, occasionally the pupillary margin is thrown aslant by it . . . It is certain that a puncture of the bulging iris with a broad needle, opening a communication with the anterior chamber, usually suffices to relieve the heightened tension, and to cause a subsidence of the rising inflammation.

Since the original description by Bowman, cases of this type have been occasionally reported in which the glaucoma has been overcome by means of needling,[2] by transfixion of the iris [3] or by iridectomy.[4]

Cases 1, 2 and 3, described in detail in the appended case reports, illustrate type 1, block of the pupil by a membrane.

In case 1 a linear extraction had been done. The pupil was relatively large, since both an iridectomy above and an iridotomy below had been done. There

1. Bowman, W.: On Extraction of Cataract by a Traction-Instrument with Iridectomy, with Remarks on Capsular Obstructions and Their Treatment, Ophth. Hosp. Rep., London **4**:332, 1863-1865.

2. (*a*) Smith, P.: On the Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891. (*b*) Chance, B.: Glaucoma After the Extraction of Cataract, Ophthalmology **6**:565, 1909-1910.

3. Fuchs, E.: Ueber Transfixion der Iris, Ber. ü. d. Versamml. d. ophth. Gesellsch. **25**:179, 1896. Lowenstein, A.: Lanzentransfixion, Ztschr. f. Augenh. **67**:68, 1929.

4. (*a*) Rumszewicz, K.: Zur Casuistik des Glaucoms nach Staar-Operationen, Klin. Monatsbl. f. Augenh. **34**:191, 1896. (*b*) Weeks, J. E.: Glaucoma After Cataract Extraction, Tr. Am. Acad. Ophth. **28**:162, 1923. (*c*) Piekarska-Miaczynska, M.: Ueber das Verhalten des intraokularen Druckes an staroperierten Augen, Klin. oczna **13**:283, 1934. (*d*) Risley, S. D.: Glaucoma in an Aphakial Eye Three Years After Extraction, Am. J. Ophth. **16**:328, 1899. (*e*) Smith.[2a]

was a very thin inflammatory membrane in the pupil. The anterior chamber became steadily shallower as the tension rose, but it was a uniform shallowing and did not at all suggest iris bombé. Under treatment with 2 per cent pilocarpine nitrate and 1 per cent epinephrine hydrochloride, the tension increased from 36 to 48 mm., but this was immediately and permanently relieved, and the anterior chamber deepened, with a simple incision of the membrane.

In case 2 the lens substance had been absorbed after needling in early childhood. The pupil was 3 to 4 mm. in size. During the course of active iridocyclitis the anterior chamber became extremely shallow without iris bombé, and tension, which had previously been normal, rose rapidly to 45 mm. The pupil was filled with a membrane. After discission of the pupillary membrane, the anterior chamber became deep and tension normal. Three months later the anterior chamber again became quite shallow, and this time typical iris bombé was noted. The pupil was again completely occluded by a membrane, and tension was 40 mm. An iridotomy and division of the membrane were done with a knife needle. The next day the anterior chamber was deep and tension normal. When the patient was last seen, twelve months later, there was an adequate pupillary opening, and tension was normal.

A third instance of pupillary block by a membrane is seen in the right eye in case 14. The patient had a mature cataract in the right eye, and acute glaucoma developed, evidently secondary to swelling of the lens. The tension dropped to normal with the use of strong miotics; and eight days after his admission, when the eye was almost quiet, an uneventful intracapsular cataract extraction with iridectomy was done. A violent fibrinous iritis developed within a few days after the operation; it gradually quieted down, leaving a dense inflammatory membrane completely filling the pupil. The pupil became somewhat drawn up. Two months after operation there was no active iritis, although the eye was still slightly congested. The anterior chamber was shallow, and tension, which had previously been rather low, was 34 mm. A double transfixion[5] of the iris was done near the periphery below. The anterior chamber deepened at once and remained deep, and tension has been normal to date without medication. A needling of the membrane will have to be carried out later.

In the first 2 cases needling of the membrane proved completely effective. Such a procedure is not entirely without risk, however, for if normal vitreous lies just behind the membrane it may press forward through the opening and recreate a pupillary block. In cases 1 and 2 there had been long-standing iritis or uveitis, and quite possibly fluid vitreous. In the third case it was considered wiser to reestablish communication between the chambers by means of transfixion of the iris

5. Transfixion is done with a broad cataract knife as near the periphery of the iris as possible. The knife is passed through the cornea, and the tip is engaged in the iris and passed through it, keeping the point parallel with the posterior surface of the iris. It can then be pushed back through the iris into the anterior chamber, thus making a second hole in the iris. One should exercise great care to avoid plunging the point of the knife backward into the vitreous, for if this is done the purpose of the operation might be defeated by the openings in the iris becoming blocked by vitreous. Should this occur, a small peripheral iridectomy could be done in another place. Of course, if the iris is in bombé form, the transfixion is easily done, without danger of injuring the vitreous.

before needling the pupillary membrane, in the hope of preventing pupillary block with vitreous when discission is done.

TYPE 2.—*Aphakic eyes, nearly always without iridectomy, in which the pupil is blocked by a herniation of vitreous through it, usually after discission of secondary cataract.*

In most of the cases of this type the glaucoma has followed needling of a membrane after simple extraction, but a few cases have been reported in which a combined extraction had been done.[6] There is often typical iris bombé. Bowman [1] recommended transfixion of the iris for this type. Von Graefe [7] was evidently well aware of the danger of this type of secondary glaucoma after needling, for he stated, "One should never do a discission in the presence of a small pupil." Hudson [8] in 1910, in a brilliant paper, was the first to suggest that the cause of the glaucoma of this type is a blocking of the pupil with vitreous. He made his deductions entirely from examination of enucleated eyes. He found in all cases of this type, in which the eyes were enucleated for intractable glaucoma after needling, that the iris was in bombé form and that the pupil was completely filled with vitreous, which herniated into the anterior chamber. He noted many adhesions between the iris and the vitreous. He recommended transfixion of the iris, as first advocated by Bowman,[1] as the treatment of choice in the early stages and iridectomy, as first advocated by H. Knapp,[9] in the later stages. In other reports on this type of glaucoma, transfixion of the iris [10] and iridectomy [11] have proved equally successful. Doubtless because cases of the type under discussion have been overlooked by us, we are able to present only 3 examples from our own experience. These cases, and 3 important cases from the literature, are given in detail in the appended case reports.

CASE 3 (Urbanek [12]).—After a wide discission of a secondary cataract which developed after linear extraction, the tension of the eye became elevated and the

6. Ascheim, H.: Die Transfixion der Iris, Arch. f. Augenh. **37**:117, 1898. Dalén, A.: Ueber Glaukom nach Starextraktion, Mitt. a. d. Augenklin. d. Carolin. med.-chir. Inst. zu Stockholm, 1901, no. 3, p. 75.

7. von Graefe, A.: Beiträge zur Pathologie und Therapie des Glaucoms, Arch. f. Ophth. **15** (pt. 3):224, 1869.

8. Hudson, A. C.: Injury to the Vitreous Body as a Factor in the Production of Secondary Glaucoma, Roy. London Ophth. Hosp. Rep. **18**:203, 1911.

9. Knapp, H.: Ueber Glaucom nach Discission des Nachstaars und seine Heilung, Arch. f. Augenh. **30**:1, 1895.

10. Teale, T. P.: Puncture of the Cornea for the Relief of Tension, Brit. M. J. **1**:404, 1864. Lichtenburg, J. S.: Glaucoma Following Cataract Operation, J. Missouri M. A. **19**:345, 1922. Footnote 6.

11. Mitchell, S.: Two Cases of Glaucoma Following Cataract Extraction, Ophth. Rec. **6**:507, 1897. von Hippel, A.: Ueber die dauernden Erfolge der Myopieoperation, Arch. f. Ophth. **49**:387, 1900. Rumszewicz.[4a] Knapp.[9]

12. Urbanek, J.: Vorfall des Glaskörpers in die vordere Kammer als Ursache intraocularer Drucksteigerung, Ztschr. f. Augenh. **54**:164, 1925.

iris bulged forward in typical bombé form. Urbanek observed with the slit lamp that vitreous completely filled the pupil and herniated forward so as almost to reach the cornea. After transfixion, the iris rapidly resumed its normal position; tension became normal and remained so during a two year period of observation. Shortly after transfixion of the iris, the vitreous was noted to withdraw somewhat from the anterior chamber, and when the patient was examined two years later vitreous filled only a part of the pupil and projected very little into the anterior chamber. The hole in the iris was still patent. It was Urbanek's opinion that the constant motion of the pupil in expansion and contraction, as well as its undulating motion, had allowed a slight seepage of aqueous, which accounted for the relatively slow development of the glaucoma and the fact that the tension never reached a very high level. He expressed the belief that normal vitreous, at least in certain circumstances, was relatively impermeable to aqueous and could therefore act as a mechanical plug to the pupil, as in the case reported. The large herniation of vitreous into the anterior chamber he believed was explained as follows: The greater pressure in the posterior chamber forced more vitreous into the anterior chamber each time a rhythmic, slight dilatation of the pupil took place, and the herniation of vitreous was gripped and held there by the following pupillary contraction. Thus, more and more vitreous tended to enter the anterior chamber. He noted no vitreous near the angle of the anterior chamber at any time in the case reported.

Cases 4 and 5 (Pereira [13]).—These cases present especially interesting features. In case 4 glaucoma followed discission and presented the same features as those in Urbanek's case. Pereira also observed with the slit lamp that the relatively small pupil was completely filled with vitreous and that vitreous herniated to a considerable extent into the anterior chamber. The iris bulged forward. Cyclodialysis was completely ineffective. Under treatment with atropine the tension gradually fell to normal; but when atropine was withdrawn the iris again bulged forward and the glaucoma returned. The hypertension was permanently relieved by transfixion of the iris. He, like Urbanek, stated the opinion that normal vitreous is relatively impermeable to aqueous.

Case 5 was somewhat different. The patient had had an intracapsular extraction with peripheral iridectomy, but the pupil became drawn up, "presumably from vitreous being caught in the wound." Five weeks after operation the eye became red and the vision blurred. The anterior chamber was very shallow and the tension elevated. Examination with the slit lamp revealed vitreous bulging through the pupil almost to the cornea. With instillation of atropine, the tension became lower, and the vitreous largely withdrew from the anterior chamber. Two months later, after strenuous bodily exertion, the original condition recurred. Pereira assumed that during the strenuous bodily exertion a dilatation of the pupil occurred, which allowed vitreous again to come forward into the anterior chamber, and that this herniation completely filled the pupil when it again contracted. On this occasion atropine failed to relieve the condition, and two posterior sclerotomies likewise failed. Finally, an iridectomy was done, after which the anterior chamber again became deep, and tension was permanently controlled, although there still remained some vitreous herniating into the anterior chamber.

13. Pereira, R. F.: Hipertensión ocular por prolapso del cuerpo vítreo en la cámara anterior después de la discisión de la catarata secundaria, Arch. de oftal. de Buenos Aires **9**:115, 1934; Prolapso del vítreo en la cámara anterior y glaucoma después de la extracción intracapsular de la catarata, ibid. **11**:409, 1936.

CASE 6.—The patient had had an uneventful intracapsular extraction with iridectomy. Convalescence was apparently uneventful except for mild postoperative iritis. Five months after operation the eye still showed a mild iritis, with the pupil occluded by an inflammatory membrane. Five months later tension was 24 mm., and there was a suggestion of forward bulging of the iris below. A wide discission was done. The following day tension was 56 mm. and there was iris bombé. The iris was transfixed, and a small subconjunctival incision in the sclera was made. There was considerable hemorrhage into the anterior chamber, which in a few days necessitated paracentesis and irrigation of the anterior chamber on account of increased tension. Thereafter the tension remained normal and the iris resumed its normal contour. Five days after the paracentesis it was noted that vitreous was bulging through the pupil, filling the upper half of the anterior chamber. The eye gradually quieted down and had vision of 20/40 with correction. The patient has been followed for five years and has had no further trouble, the tension always remaining normal. There is no further note in the record relative to the presence or absence of vitreous in the anterior chamber.

CASE 7.[14]—The patient had had severe uveitis and had many posterior synechias. There was an immature cataract. Tension began to rise, and an intracapsular extraction with broad basal iridectomy was done. After several months the tension became elevated and was controlled for some time with miotics, but eventually a cyclodialysis had to be done. Shortly after operation it was noted that an inflammatory membrane completely filled the pupil and the coloboma except for a space about 2 mm. in diameter at the lower border of the pupil. Vitreous began to herniate through this small opening, and eight days after operation vitreous was in contact with the cornea. Tension remained 13 to 16 mm. Three months later it was noted that vitreous filled about three fourths of the anterior chamber and had caused corneal edema in two places. Tension was 26 mm. A single puncture was made in the iris with a cataract knife near the limbus at 2 o'clock. The anterior chamber deepened immediately. It had never been very shallow, but not as deep as in the usual aphakic eye. On the patient's discharge, on the day following the iris puncture, the anterior chamber was deep and vitreous was nowhere in contact with the cornea. The patient has been followed for two months. Tension has remained normal and the anterior chamber deep, and vitreous has withdrawn considerably from the anterior chamber.

CASE 8.—In this case an extracapsular cataract extraction with iridectomy had been done. Convalescence had been uneventful except that there was a pupillary membrane, partly capsular, partly inflammatory. Two months after cataract extraction a discission was done and a good opening secured, with resulting vision of 20/25 with glass. Three years later the patient returned to the clinic with a history of severe ocular pain and inflammation, of six days' duration. The eye was severely injected, and the cornea was steamy. The anterior chamber was somewhat shallow, particularly on the sides, and was deeper below. The pupil was slightly drawn up, and the coloboma was rather narrow. Some of the capsule was present in the upper part of the coloboma, but there was a good opening in the lower part of the pupil, through which vitreous was herniating forward, so as to reach almost to the cornea. Tension was 52 mm. A single puncture was made in the iris with a cataract knife near the periphery at the 4 o'clock position. The anterior chamber deepened at once. A single dressing was applied and the patient allowed to return home. Two days later the eye was almost white and quiet; the vitreous had retracted to the level of the

14. This case is reported through the courtesy of Dr. Mahlon Easton.

iris, and tension was 20 mm. The patient has been followed for two months. Tension has ranged from 23 to 15 mm. The eye has remained quiet, and the vitreous has stayed on the plane of the iris. This case is somewhat unusual in that the block of the pupil with vitreous did not occur until three years after the discission, and without any obvious cause.

TYPE 3.—*Eyes in which after intracapsular lens extraction the pupil is blocked by adhesion of the iris to the intact hyaloid membrane.*

We have been unable to find any cases of this type reported in the literature. Cases, 9, 10, 11 and 12 are reported in detail in the appended case reports. Unfortunately, some of the pertinent details are lacking in the records, but the general pictures are fairly complete.

CASE 9.—In this case, one month after an uneventful intracapsular extraction through the round pupil with a small peripheral iridotomy, it was noted that the anterior chamber was quite shallow, but tension was normal. The eye remained somewhat congested. Two weeks later the anterior chamber was still very shallow, and tension was 48 mm. Four per cent pilocarpine nitrate, given every two hours, failed to reduce the tension. The hyaloid membrane bulged slightly through the pupil but was intact. It was noted that the small iridotomy opening seemed to be closed by a membrane. A cyclodialysis failed to relieve the condition. Three weeks later a trephine was done, and the record states that "a small iridotomy was done, and there was some loss of vitreous." Though there is no note in the record to that effect, it is possible that this iridotomy opening was plugged with vitreous. At any rate, the anterior chamber remained shallow, and three days after the trephine operation the tension was 36 mm. During the next two weeks the tension remained elevated. Then cyclodiathermy of the lower half of the globe and posterior sclerotomy in the lower outer quadrant were done. For three weeks, with massage and therapy with miotic drugs, the tension remained normal as determined by palpation and then rose to 36 mm. The anterior chamber remained very shallow. The pupil was 2 to 3 mm. in diameter and did not react to light. At this time it occurred to one of us for the first time that the glaucoma might be due to a block between the posterior and the anterior chamber. A small, single puncture in the iris was made. Two days later it was noted that the anterior chamber was definitely deeper and the tension was normal to palpation, but there was an anterior synechia at the site of the puncture, and the hole in the iris seemed to be blocked with blood and exudate. One week after the iris puncture the anterior chamber was again very shallow and the tension was 45 mm. At this time the true nature of the situation was finally recognized. Since the anterior chamber had been shallow for so long, cyclodialysis with basal iridectomy was done. There was no loss of vitreous at operation. On the day following operation the anterior chamber was deep and tension normal. The patient has been under observation for three and a half months since the last operation. The tension has ranged from "mushy soft" to 14 mm., as recorded at the last examination. Doubtless the cyclodiathermy, as well as the cyclodialysis done at the time of the iridectomy, contributed to the relative hypotony; but it is clear from the prompt and permanent deepening of the anterior chamber after the iridectomy that the decisive factor in the final relief of the glaucoma was the reestablishment of free communication between the posterior and the anterior chamber, since cyclodiathermy combined with posterior sclerotomy, and cyclodialysis had previously failed to deepen the anterior chamber or to control the tension. At the last examination it was noted that there was complete posterior

synechia between the iris and the hyaloid membrane, except in the iridectomy coloboma. This adhesion had evidently completely sealed off the posterior from the anterior chamber. In retrospect, it is probable that if at the first sign of glaucoma an adequate transfixion of the iris had been done the condition might have been relieved at the outset and the patient spared a series of fruitless operations.

CASE 10.—This case presents somewhat different features. Here, there were a widely dilated, fixed pupil from trauma and an iridectomy upward. In spite of gross loss of vitreous at the time of extraction of the lens, there was never any free vitreous in the anterior chamber after operation. What appeared to be a smooth, intact hyaloid membrane stretched across the pupil and was everywhere in contact with the pupillary margin as far up into the coloboma as could be seen. Fine posterior synechias were present. The anterior chamber was always shallow after extraction of the lens and gradually became more so, until five weeks after operation it was almost flat. The tension steadily rose under treatment with miotics and epinephrine. When iridectomy was done, no vitreous was lost; yet the anterior chamber became generally deep and has remained so, except that the small segment of iris remaining between 1 and 3 o'clock was apparently firmly adherent to the back of the cornea. After iridectomy the hyaloid membrane pulled away somewhat from the iris, and it could be seen that the synechia had been complete. The anterior chamber had remained shallow so long that the angle is doubtless permanently closed in part, and further surgical intervention may be necessary to control the glaucoma. However, the marked reduction in tension and the deepening of the anterior chamber after iridectomy indicate that here, also, there existed a block between the anterior and the posterior chamber.

CASE 11.—In this case, after an uneventful intracapsular cataract extraction with iridectomy, the patient was discharged from the hospital with a normal anterior chamber and with normal tension. When he was next seen, about six weeks later, the eye was considerably injected and the anterior chamber was deep. Three days later the anterior chamber was shallow and the tension 35 mm. A 2 per cent solution of pilocarpine nitrate was given, but three days afterward the anterior chamber was extremely shallow and the tension 48 mm. In an attempt to do a paracentesis, very little aqueous escaped, but the iris prolapsed through the paracentesis opening. When an attempt was made to reposit it, a small hole was made in the iris. At this point there was escape of more fluid, and the anterior chamber deepened at once. Thereafter the anterior chamber remained deep; tension was normal, and the eye gradually quieted down. The hole inadvertently made in the iris evidently drained the posterior chamber and reestablished communication between the anterior and the posterior chamber. There is no note in the record of the relation of the pupil to the hyaloid membrane at the time of operation, but subsequently a thin inflammatory membrane in the pupil and posterior synechias were noted. It is probable that the inflammation of the iris had sealed the iris to the hyaloid membrane so as completely to block the pupil.

In the last 3 cases discussed, the true nature of the situation was sooner or later recognized and proper measures were taken, albeit in case 11 accidentally. In case 12, however, the true nature of the process was never recognized.

CASE 12.—In the beginning, the situation in case 12 was exactly the same as that in case 9. Intracapsular extraction had been done, with a small peripheral iridotomy. Gradual shallowing of the anterior chamber and onset of glaucoma

followed, the eye remaining always irritable. Many times it was noted that the pupil was still small and did not react to light. Then followed a long series of operations: A subconjunctival keratome incision into the anterior chamber, done twice; posterior sclerotomy; cyclodialysis; trephination; paracentesis, and cyclodiathermy, done twice. When the patient was last seen, the tension was 42 mm., and vision equaled 3/200.

In the beginning of the glaucoma in this case it was noted that the pupil was small and immobile. In the light of the other cases reported, it is probable that the pupil was blocked by adhesions between the iris and the hyaloid membrane. It should be noted that in none of these 4 cases did the iris take on the typical bombé form. There was a general uniform shallowing of the anterior chamber. In none was there a herniation of vitreous through the pupil. At most there was a slight forward bulge of the intact hyaloid membrane into the pupil. It is probable that there must be considerable postoperative inflammation in these cases to seal the iris to the hyaloid membrane and that this could be avoided if the pupil were kept dilated. All these eyes were noted to be irritable for some time after operation. The small peripheral iridotomy openings in cases 9 and 12 were evidently blocked by inflammatory membrane or by vitreous.

TYPE 4.—*Eyes with partial or complete subluxation of the lens backward, in which the pupil is blocked by a herniation of vitreous through it.*

A great many reports of glaucoma due to a dislocated lens are to be found in the literature, but the role of the vitreous acting as a block of the pupil in these cases has apparently not hitherto been clinically recognized, though Hudson,[8] reporting on the pathologic examination of enucleated eyes, correctly deduced the mechanism of the glaucoma. He examined 2 eyes that had been enucleated for glaucoma and noted in each a subluxation of the lens with herniation of the vitreous through the pupil. The iris was everywhere pushed forward so as to block the angle, but the block of the angle was most complete on the side of backward subluxation of the lens. The clinical details of these cases were not available, but he concluded that the glaucoma must have been caused by the pupil being blocked by vitreous, this resulting ultimately in a flat anterior chamber and complete block of the angle.

Another of Hudson's cases was that of a woman aged 43 who had a severe contusion of one eye. On examination nine days later, the pupil was dilated, the lens was completely dislocated into the posterior chamber, the iris was in bombé form and the tension was elevated. Two paracenteses and a cyclodialysis failing to relieve the tension, the eye was enucleated. Pathologic examination showed iris bombé, with the angle completely closed. Vitreous completely filled the pupil and herniated forward into the anterior chamber. The vitreous was everywhere in close contact with the pupillary margin and showed numerous adhe-

sions here. Hudson stated the belief that in this, as in his other, cases the glaucoma had been due to the pupil being blocked by vitreous. Cases 13 and 14, reported in detail in the appended case reports, illustrate this type.

CASE 13.—In this case, the upper nasal border of a hypermature cataract was subluxated backward, and vitreous was presenting in the pupil between the iris and the lens. There was prominent exfoliation of the lens capsule. On the patient's first admission, the anterior chamber was observed to be deep except in the lower outer quadrant, where it was shallow peripherally, evidently from a forward tilting of the lens here. Tension was 42 mm. Presumably, the increased tension was due to a partial closure of the angle in the lower outer quadrant and to the capsular exfoliation, since it quickly responded to treatment with 4 per cent pilocarpine nitrate and 0.5 per cent physostigmine salicylate. On his second admission, twelve days later, the anterior chamber was almost flat, but the shallowing was uniform; there was no sign of iris bombé. The lens was seen to be well back of the plane of the iris. Tension was 48 mm., and the eye was extremely injected and painful. Owing to the edematous cornea and the extreme shallowness of the anterior chamber, it was not possible to identify vitreous in the pupil; but it was assumed that vitreous must be present and acting as a plug. When transfixion of the iris was done, aqueous gushed out along the knife blade even before it was withdrawn from the eye; the anterior chamber at once became deep and has remained so. The eye rapidly whitened, and tension remained normal without treatment until three weeks later, when it was found to be 30 mm. The holes in the iris made by the transfixion were patent; the anterior chamber was everywhere deep except, as before, in the periphery of the lower outer quadrant. Some vitreous was still present in the pupil. Pilocarpine was prescribed, and since that time tension has been under 30 mm. when the patient has used the miotic faithfully.

CASE 14.—In this case the lens was completely dislocated backward, and the cause of the glaucoma was less clear. The anterior chamber was slightly shallow, about the same as in the other eye, in which the lens was in situ. It was somewhat shallower than in the usual aphakic eye. With the slit lamp it could be seen that vitreous was in the pupil and that it practically filled the anterior chamber. It was thought at first that the cause of the glaucoma was probably a mechanical block of the angle by vitreous. The tension, however, had risen from 25 to 57 mm. in two weeks under miotic therapy. It seemed possible, therefore, that a block of the pupil by vitreous might be a factor in the glaucoma, especially since the anterior chamber was not as deep as one would expect in such an eye. A relative increase in pressure in the posterior chamber from a blocked pupil would also explain the large amount of vitreous in the anterior chamber. It was decided, therefore, to do a transfixion of the iris, at least, as a preliminary measure. When this was done, the anterior chamber deepened at once. When air was introduced into the anterior chamber and later allowed to escape, the vitreous following was found to be of normal consistency. Postoperatively atropine was given, but this dilated the pupil only to a diameter of 5 mm. The anterior chamber remained deep and the tension normal until two weeks after the operation, when a tension of 30 mm. was found. A miotic was prescribed, which has controlled the tension to date. Two weeks after the transfixion of the iris it was noted that there was no vitreous in the anterior chamber, except one strand, 2 to 3 mm. in diameter, extending through the pupil and up to the corneal wound. One week later only a very narrow strand, not more than 1 mm.

in diameter, remained. It would seem, therefore, that the mechanism of the glaucoma in this case, as in case 13, was a block of the pupil by vitreous. The block probably developed more gradually than in case 13 and never became complete. Such an assumption would explain the more gradual onset of the glaucoma, the large amount of vitreous in the anterior chamber, and the fact that the anterior chamber never became extremely shallow.

COMMENT

In all four types of cases the shallowing of the anterior chamber may ultimately result in mechanical closure of the angle. Hence it is important that the proper treatment be instituted early, before permanent adhesions are formed. If one excepts cases of shallow or flat anterior chamber, due to leakage at the wound, there seems to be no cause for progressive shallowing of the anterior chamber in an aphakic eye, or one in which the lens is partially or completely dislocated backward, other than lack of free communication between the posterior and the anterior chamber. Typical iris bombé, when present, at once suggests a blocked pupil. However, in cases in which there is no such condition, but only a general, or uneven, shallowing of the anterior chamber, as in several cases already discussed, the true situation is not so obvious.

Furthermore, a dense pupillary membrane may have such firm adherence to the iris peripheral to the pupillary border as to prevent a general shallowing of the anterior chamber. In such doubtful cases a transfixion of the iris can be carried out as a diagnostic procedure. Case 6 illustrates the hazard of a wide discission in the presence of a completely blocked pupil and increased tension, in causing a marked herniation of vitreous into the anterior chamber and a block of the pupil by vitreous. A preliminary transfixion of the iris in this case, such as was done in the right eye in case 14, would have equalized the pressures in the posterior and anterior chambers and would in all probability have prevented the marked herniation of vitreous into the anterior chamber following the discission, with its unfortunate consequences. If a pupil is completely blocked with a membrane partly or wholly inflammatory, it would seem wise always to precede the discission by a peripheral transfixion of the iris.

In cases of subluxation of the lens, the glaucoma may often be due to a tilting forward of one side of the lens, pressing the iris forward here and blocking the angle, since peripheral anterior synechias thus formed in one place may spread around the periphery until enough of the angle is closed to cause glaucoma. Smith [2a] and Lawford,[15] among others, have made pathologic reports on such eyes. Except for the pathologic observations of Hudson,[8] already referred to, the possibility of a block of

15. Lawford, J. B.: Cases of Dislocation of Crystalline Lens, Ophth. Hosp. Rep., London **11**:327, 1886-1887.

the pupil by vitreous in some of these cases has apparently never previously been considered. In most of the reports the presence or absence of vitreous in the pupil is not recorded, and often the depth of the anterior chamber is not noted. Hegner,[16] however, without recognizing the importance of his observations, noted in several of his cases a generally shallow or flat anterior chamber, and in some he observed vitreous occupying the space between the lens and the edge of the pupil. The observations in his cases 8, 9 and 12 bear a striking resemblance to those in our case 13. In his cases 8 and 9 the eye was enucleated, and in case 12 vision was reduced to light perception from glaucoma. It is probable that in these cases, as in many others reported in the literature, a block of the pupil by vitreous was the essential factor in producing the glaucoma.

In cases of complete luxation of the lens backward, shallowing of the anterior chamber on one side when the lens lies against the ciliary body and iris may possibly be a factor in the glaucoma, as in case 1 reported by Lawford.[15] In most other cases reported, however, it has been assumed that the cause of the glaucoma was increased secretion from the ciliary body, due to irritation by the lens; hence, lens extraction has been almost universally employed in treatment. Evidence is lacking, however, that such hypersecretion actually takes place. In the majority of cases of posterior luxation the lens is well tolerated in the vitreous chamber, and no glaucoma occurs. Indeed, Suker[17] reported a case of posterior luxation of the lens of thirty years' duration, with normal tension and preservation of 20/40 vision.

In Lawford's[15] case 3 the patient had had no trouble with the eye for twelve years after the lens became dislocated; then intractable glaucoma developed and the eye had to be enucleated. Pathologic examination showed a deep anterior chamber and an open angle. The anterior chamber was filled with a "soft semi-transparent effusion." The lens lay on the retina posterior to the ora serrata. There was an aggregation of cells in the meshes of the pectinate ligament and about Schlemm's canal, which the author thought might retard the outflow of aqueous. One wonders whether the "coagulated, soft semi-transparent effusion" in the anterior chamber, which microscopically consisted of a "very fine fibre-network with some faintly granular material," was not vitreous, and whether its presence in the anterior chamber was not the cause of the glaucoma. In Hegner's[16] case 11, of posterior dislocation, there were at first a flat anterior chamber and a tension of 70 mm.

16. Hegner, C. A.: Klinische Untersuchungen über die Prognose der intrabulbären Linseluxationen, Beitr. z. Augenh. **9**:707, 1914-1915.

17. Suker, G. F.: A Dislocated Lens in the Vitreous of Thirty Years Standing in One Eye, Ophth. Rec., 1902, p. 462.

Examination some time later revealed that the anterior chamber was deep and that vitreous was herniating through the pupil into the anterior chamber. Tension was 43 mm. It may be that in our case 14 a shallow anterior chamber had preceded the marked herniation of vitreous into the chamber. Possibly, in the later stages the important factor in the glaucoma is mechanical block of the angle by vitreous, which has been forced into the anterior chamber by increased pressure in the posterior chamber as a consequence of pupillary block by vitreous.

After intracapsular extraction one frequently observes some herniation of vitreous through the pupil, evidently due to lack of support by a weak hyaloid membrane. Such prolapse of the vitreous usually does not completely fill the pupil and shows little tendency to continue to advance into the chamber. If, however, the pupil is completely blocked by vitreous and there is no free communication between the chambers, such as is afforded by a patent peripheral iridectomy opening, one may observe a progressive accumulation of vitreous in the anterior chamber to the point of almost completely filling it, with resulting glaucoma.

When free communication is reestablished between the chambers as a result of transfixion of the iris or iridectomy, there is apparently a strong tendency for the vitreous to withdraw from the anterior chamber, as illustrated in cases 3, 4, 5, 7, 8 and 14. Therefore, transfixion of the iris or iridectomy would seem to be indicated, even if the anterior chamber is deep, if it is filled with vitreous. In fact, the withdrawal of vitreous from the anterior chamber as a result of transfixion of the iris or iridectomy may explain the relief of glaucoma in cases in which obstruction to the outflow of aqueous has been due to vitreous in the angle rather than to closure of the angle by forward displacement of the iris. That vitreous filling the pupil offers sufficient obstruction to the flow of aqueous to cause glaucoma is proved, in our opinion, by the dramatic fall in tension which follows transfixion of the iris or iridectomy.

Whatever other causes for glaucoma may operate in cases of backward dislocation of the lens, it seems certain, in the light of Hudson's pathologic report and our clinical observations, that a block of the pupil by vitreous must often be a factor. The presence or absence of vitreous in the pupil or in the anterior chamber can easily be verified by examination with the slit lamp. If vitreous is seen to be herniating through the pupil and the tension is elevated, a transfixion of the iris should be carried out, at least as a preliminary measure. In eyes previously treated by iridectomy and removal of the lens from the vitreous, with relief of the glaucoma, it is possible that the important factor was the iridectomy, which reestablished free communication between the chambers, and not the removal of the lens per se.

SUMMARY

A form of secondary glaucoma in aphakic eyes and in eyes with dislocated lenses is discussed, in which the causative factor is apparently a block between the anterior and the posterior chamber.

This block may be from a membrane partly or wholly inflammatory, from a herniation of vitreous through the pupil acting as a plug or from adhesion of the iris to an intact hyaloid membrane.

Treatment should be directed toward reestablishment of communication between the chambers by the use of a mydriatic, by discission of a pupillary membrane, by transfixion of the iris or by iridectomy.

It is important that proper treatment be undertaken early, before permanent peripheral anterior synechias are formed.

A block of the pupil by vitreous in an aphakic eye, or one with posterior dislocation of the lens, with resultant increased pressure in the posterior chamber, is the probable cause of large accumulations of vitreous in the anterior chamber.

In an aphakic eye, or in one with a backward dislocation of the lens, the development of iris bombé, a general or uneven shallowing of the anterior chamber or an increasing accumulation of vitreous in the anterior chamber probably indicates that there is a block between the chambers and that glaucoma is imminent, if it is not already present.

In aphakic eyes in which the pupil is completely blocked by a membrane, partly or wholly inflammatory, it is advisable to do a preliminary transfixion of the iris before undertaking a discission of the membrane.

In view of our experience in cases 13 and 14, it is probable that the heretofore not clinically recognized block of the pupil by vitreous in eyes with posterior dislocation of the lens is the chief cause of the glaucoma and that in many cases of this type which have been reported in the literature the glaucoma could have been relieved by transfixion of the iris or by iridectomy.

REPORT OF CASES

CASE 1.—F. S., a girl aged 13.

May 29, 1939.—There was a history of arthritis and of chronic inflammation in both eyes for four years, with gradual loss of vision. Examination revealed low grade uveitis in both eyes, with many posterior synechias and some pupillary membrane, early band keratitis and immature cataract; iris bombé was present in the left eye. Vision was 1/200 in the right eye and 20/100 in the left eye. Tension was 17 mm. in the right eye and 30 mm. in the left eye.

June 28.—Iridectomy was done on the left eye.

March 22, 1942.—The patient was admitted for extraction of the lens of the left eye. Each eye showed moderate band keratitis, a normal anterior chamber, many old posterior synechias, a pupillary membrane and mature cataract. In the left eye there was a coloboma of the iris upward from a previous iridectomy. (Examination with the slit lamp revealed no cells in the aqueous of either eye

and no precipitates on the back of the cornea.) Tension was normal to palpation in both eyes. Vision was ability to see shadows, with accurate light projection, in each eye.

March 3.—A linear cataract extraction with iridectomy upward and inferior iridotomy was performed on the left eye. After a moderately stormy convalescence, the eye became quiet, with a clear pupil, and vision with a lens equaled 20/20—. This vision has been maintained, and there has been no further trouble with this eye.

September 22.—Lens extraction with a small iridectomy upward and inferior iridotomy was performed on the right eye. There were some postoperative hyphemia and moderate inflammation. Atropine was used continuously after the operation.

October 19.—Vision was 20/70 in the right eye with a + 10.00 D. sphere and a pinhole disk. There was a thin pupillary membrane; the anterior chamber was very shallow, and tension was 36 mm. Use of atropine was discontinued, and 2 per cent pilocarpine nitrate and 1 per cent epinephrine hydrochloride were prescribed.

October 26.—Vision was 20/50 + 2 in the right eye with a + 10.00 D. sphere and a pinhole disk. The eye was moderately injected, with slight corneal edema, a very shallow anterior chamber and tension of 45 mm. Admission to the hospital was advised.

November 2.—The patient was admitted to the hospital. Tension was 48 mm. in the right eye.

November 4.—With a Ziegler knife, the pupillary membrane was divided with a single vertical incision. The next day the anterior chamber was deep and tension was normal. Since that time tension has ranged from 10 to 18 mm., without medication.

Subsequent Course.—The patient was last seen on Jan. 14, 1946. Tension was then 18 mm. in the right eye, and vision in that eye with glass was 20/20—.

CASE 2.—O. F., a woman aged 23.

Jan. 10, 1941.—There was a history of congenital cataract of both eyes and several needling operations in early childhood. The left eye was convergent; the anterior chambers were of normal depth, and the pupils were slightly irregular and 3 to 4 mm. in diameter, with good central openings. Tension in both eyes was normal to palpation. Vision with glass was 20/200 in the right eye and 4/200 in the left eye.

April 30, 1943.—The right eye was quiet, with many precipitates on the back of the cornea, a thin pupillary membrane and opacities in the vitreous.

Feb. 18, 1944.—The right eye showed many keratic precipitates.

October 18.—The right eye was considerably injected; the anterior chamber was of normal depth; the pupil was small and nearly filled with inflammatory membrane. Tension was normal to palpation.

November 21.—The patient was admitted to the medical ward with a questionable diagnosis of rheumatic fever. While she was in the ward the right eye remained inflamed. Treatment consisted of instillations of atropine and application of hot compresses. On account of the medical condition, foreign protein and salicylates were not given.

December 11.—The patient began to suffer acute pain in the right eye. Examination showed an extremely shallow anterior chamber. Tension was 45 mm. A discission of the pupillary membrane was done and a small opening secured. The following day the anterior chamber was normally deep and tension was

normal. She was discharged from the hospital on Jan. 4, 1945, with the eye still somewhat inflamed but with normal tension.

March 20, 1945.—The right eye was slightly injected; the anterior chamber was very shallow; the iris was of bombé form, and the pupil was occluded with inflammatory membrane. Tension was 36 mm.

March 21.—With a Ziegler knife, an iridotomy and division of the pupillary membrane were done. At the time of discharge, on March 27, the anterior chamber was normal in depth except in the region of a few peripheral anterior synechias. Tension was normal.

April 10.—Tension was 15 mm. in each eye.

June 8.—Tension was normal to palpation.

Subsequent Course.—The patient was last seen on Jan. 18, 1946. Vision with glass was 18/200 in the right eye. The pupillary opening was good; the anterior chamber was normal in depth, and there were several peripheral anterior synechias. Tension was 18 mm.

CASE 3.—F. M., a youth aged 17 (Urbanek [12]).

Oct. 26, 1921.—Right Eye: The anterior chamber was normal; congenital zonular cataract was present; vision was 6/24.

Left Eye: The anterior chamber was deeper than that of the right eye. The pupil was slightly oval and smaller than the pupil in the right eye. A pupillary membrane was present. Vision was limited to counting fingers at 1 meter.

October 28.—Operation was performed on the left eye. With two needles, a wide opening was made in the pupillary membrane.

October 29.—A wide central pupillary opening was present in the left eye.

November 1.—The patient was discharged from the hospital. At the time of discharge the eye was slightly injected; the anterior chamber was fairly deep in the pupillary zone, but in the periphery it was slightly shallower than before operation. The pupil was round and slightly more than half dilated, with a wide opening and vitreous bulging through the pupil into the anterior chamber. Vision was 6/12 with glass.

November 16.—The patient returned to the clinic with pronounced ciliary injection of the left eye. The cornea was cloudy. He had been suffering some pain for several days. Tension to palpation was slightly elevated. There was iris bombé. Examination with the slit lamp showed a large herniation of vitreous into the anterior chamber, reaching almost to the cornea in the central zone. The vitreous completely filled the pupil, and the pupillary border of the iris seemed everywhere in close contact with it. The angle of the chamber appeared to be free. Tension was 35 mm. Vision with glass was 6/18.

November 18.—A transfixion of the iris was done, and the anterior chamber deepened at once.

November 19.—The eye was white and the cornea clear.

November 20.—The anterior chamber was deep, and there was less vitreous in it.

November 27.—The contour of the iris was flat. Vitreous had almost completely withdrawn from the anterior chamber but still filled the pupil. Tension was 25 mm. in each eye. The patient was discharged.

Nov. 23, 1923.—The patient was reexamined. He had had no further trouble with the eye. The pupil was slightly oval horizontally and reacted well in the upper and lower portions. There was no adhesion anywhere between the iris and

the vitreous. The hole in the iris at the site of the transfixion was patent. The fundus was normal. Vision with glass was 6/12.

CASE 4.—F. J., a man aged 25 (Pereira [13]).

Aug. 10, 1925.—The patient had a history of injury to the left eye with a piece of steel while working with a hammer and chisel. The right eye was normal. Examination of the left eye showed ciliary injection. A 3 mm. perforating wound of the cornea lay just inside the limbus at 7 o'clock. The anterior chamber was shallow nasally and superiorly. The lens was opaque, with its matter bulging into the anterior chamber from a wound in the capsule. Tension was higher in this eye than in the right eye. Atropine and cold compresses were prescribed.

September 10.—Tension was normal. The lens matter was being absorbed.

April 1926.—Only a little of the lens capsule remained in the temporal part of the pupil, between 2 and 7 o'clock. The rest of the pupil was black. The fundus was normal. Vision with glass was 6/8.

1931.—A thick secondary membrane was observed in the pupil. Vision was limited to counting fingers at 1.5 meter.

April 1933.—Vision was limited to counting fingers at 5 meters.

September 1933.—The left eye was divergent. Vision with glass was limited to counting fingers at 6 meters. The fundus was not visible.

Sept. 18, 1933.—Discission was done with two needles, resulting in a wide opening in the capsule. A large herniation of the vitreous occurred into the anterior chamber. Atropine was prescribed.

September 22.—Vision with glass was 6/8.

September 30.—There was acute glaucoma in the left eye, with severe pain. The cornea was steamy; the iris was of bombé form; tension was elevated. A subconjunctival injection of 0.2 cc. of epinephrine hydrochloride (1:1,000) was given. Atropine and a 3 per cent cocaine ointment were prescribed, and the patient was placed flat in bed, lying on his back.

October 1.—Tension was still elevated. Cyclodialysis was done between the 7 and 12 o'clock position. Use of atropine was continued.

October 5.—Tension was high. Instillation of atropine was continued. The tension gradually fell.

December 10.—The patient went back to work but returned the same night with acute glaucoma in the left eye and severe pain. The eye was intensely injected; the cornea was steamy; the pupil was small; vitreous was almost in contact with the cornea, and the pupil was filled with vitreous. Iris bombé was present. With use of atropine and a cocaine-epinephrine ointment, tension was quickly reduced.

December 14.—Double transfixion of the iris was performed temporally with a Graefe knife. The anterior chamber quickly became deeper; the cornea cleared, and tension fell to normal.

December 20.—The patient was discharged. The cornea was clear. An anterior synechia was present at 3 o'clock. The anterior chamber was deep everywhere except at the site of the anterior synechias. The vitreous retracted so that it was less than half the distance from the pupil to the cornea. The iris was on a flat plane. The transfixion holes in the iris were patent.

CASE 5.—A. M., a man aged 67 (Pereira [18]).

June 15, 1931.—The patient was admitted to the hospital with a history of failing vision of one year. Examination showed nuclear cataract in each eye.

Vision was limited to counting fingers at 0.75 meter in the right eye and was 6/18 in the left eye.

June 16.—Intracapsular cataract extraction with peripheral iridectomy was performed on the right eye.

June 17.—The anterior chamber was flat; the pupil was half dilated.

June 20.—The anterior chamber was formed.

June 29.—The patient was discharged from the hospital. There was ciliary injection; the cornea was clear except for folds in Descemet's membrane; the pupil was drawn upward, so that the upper part of the iris disappeared behind the scleral overhang. There was a separation of the choroid nasally. Vision was 6/24 with glass.

July 22.—The patient was readmitted to the hospital. Two days before admission he had vomited during the night. The following morning the eye was inflamed and painful and the vision blurred. The right eye showed ciliary injection. There was uveal tissue in the temporal part of the wound. The cornea was clear, the anterior chamber deep and the pupil drawn upward and half dilated. There was a large hernia of vitreous into the anterior chamber, which reached the cornea in the central zone. Tension was + 1. Vision with glass was 6/12. The patient was kept in bed, lying on his back, and atropine was prescribed.

July 24.—There was slight injection. The vitreous had receded to the plane of the pupil. Tension was normal. Vision with glass was 6/18.

September 26.—After twelve days of hard work at cutting wood, the patient had experienced intense pain in the right eye, and it had become inflamed. Examination of the right eye showed ciliary injection and a cloudy cornea. The anterior chamber was very shallow, being absent at the periphery. The iris was congested. The pupil was dilated and drawn upward. The vitreous was herniated through the pupil, was in contact with the cornea in the central zone and reached the angle in some places. Tension was elevated. Vision with glass was limited to counting fingers at 0.5 meter. The patient was put to bed on his back and atropine prescribed.

September 28.—The condition was unchanged. Tension was 60 mm.

September 29.—A posterior sclerectomy in the inferior temporal quadrant was done, with escape of a moderate amount of fluid vitreous. The eye failed to improve, and another posterior sclerectomy was done in the inferior nasal quadrant. After this the tension fell a little, and finally an iridectomy was done. There was moderate bleeding into the anterior chamber.

October 12.—The hyphemia was absorbed. There was slight congestion. The cornea was clear except for precipitates on the posterior surface. There were cells in the aqueous. The anterior chamber was deep. Iridodonesis was present. Tension was normal. There was a large coloboma of the iris upward, through which vitreous bulged into the anterior chamber. The disk was pale, with slight excavation. Vision with glass was 6/15.

Pereira expressed the belief that during the strenuous phsyical exertion the pupil had probably become dilated and vitreous had then come forward into the anterior chamber and was strangulated there on later constriction of the pupil.

CASE 6.—S. H., a woman aged 67.

June 23, 1939.—There was immature cataract in both eyes. Vision was limited to counting fingers at 8 feet (240 cm.) in the right eye and was 20/70 with glass in the left eye. Tension was normal in both eyes.

September 30.—Combined intracapsular cataract extraction was done on the right eye. Convalescence was complicated by mild, but prolonged, iritis with membrane formation. There was no elevation in tension.

December 4.—Refraction revealed vision of 20/50 with lens in the right eye and 20/40 with lens in the left eye.

March 22, 1940.—Both eyes were white and quiet. Tension was 20 mm. in the right eye and 22 mm. in the left eye.

July 19.—Both eyes were white and quiet. The right eye showed a thin pupillary membrane with complete posterior synechia and suggestive early iris bombé. Tension was 24 mm. Vision with lens was 20/100 in the right eye.

August 10.—A wide discission of the membrane was done on the right eye. The day following operation the tension became elevated to 56 mm. and iris bombé was present.

August 11.—With a cataract knife, the iris in the right eye was transfixed, and a small subconjunctival scleral incision was made. A large hyphemia followed this procedure. Tension remained low for three days.

August 15.—Tension was 56 mm. in the right eye. Paracentesis was done, and part of the blood was removed. After this the tension fell and remained between 12 and 15 mm. until the time of the patient's discharge. The blood was gradually absorbed. It was noted that vitreous was bulging through the pupillary opening, filling the upper half of the chamber.

August 23.—The patient was discharged from the hospital.

Subsequent Course.—She was seen repeatedly until Dec. 14, 1945, and tension was found to vary from 14 to 20 mm. Vision with lens was 20/40. There was slight cupping of the disk.

CASE 7.—J. A. P., a man aged 62.

Jan. 15, 1941.—Vision was 20/50 in the right eye and 20/70 in the left eye. Posterior synechias and immature cataracts were present in both eyes. Tension was 24 mm. in the right eye and 45 mm. in the left eye. Pilocarpine nitrate, 2 per cent, was prescribed, to be used four times a day in the left eye.

January 27.—Tension was 30 mm. in the right eye and 40 mm. in the left eye. Pilocarpine nitrate, 4 per cent, was instilled once a day in the right eye and five times a day in the left eye.

February 11.—Tension was 22 mm. in the right eye and 35 mm. in the left eye. Physostigmine ointment was added to the medication, to be used each night in the left eye.

March 18.—A combined intracapsular cataract extraction was done on the left eye. (Operation was preceded by a course of typhoid vaccine.) Convalescence was uneventful.

September 8.—Tension in the right eye had again started to rise. A combined intracapsular cataract extraction was therefore performed on that eye. (Operation was again preceded by a course of typhoid vaccine.) Again, convalescence was satisfactory. Subsequent vision was 20/15 in the right eye and 20/100 in

the left eye. The poor vision in the left eye was due to glaucomatous changes. Tension in both eyes remained satisfactory until the winter of 1942, when it became slightly elevated in both eyes. Miotics controlled the tension for a time.

Feb. 2, 1943.—An acute exacerbation of glaucoma occurred in the right eye. The patient was hospitalized and treated with miotics, which reduced the tension satisfactorily.

March 2.—The patient was discharged, with instructions to use pilocarpine nitrate, 2 per cent, every three hours in the right eye and four times a day in the left eye. Since this time vision in the left eye has been between 20/100 and 20/200, with a reduced field of vision. The tension has remained satisfactory. Vision in the right eye remained 20/20, with a normal field but with borderline tension (20 to 30 mm.), until the fall of 1945.

Sept. 17, 1945.—Two days before, the patient had a strenuous day and experienced a spell of blurred vision. Tension was 29 mm. in the right eye and 22 mm. in the left eye. Treatment was continued unchanged.

November 14.—Blurring and halos were experienced in the right eye. The cornea was steamy, with a few posterior synechias. Tension was 37 mm. Furmethide (furfuryl trimethyl ammonium iodide), 1 drop, was instilled in the right eye for four doses at fifteen minute intervals. The tension was unchanged. The patient was admitted to the hospital and given an intensive course of treatment with miotics, which reduced the tension to 29 mm. Gonioscopic examination showed the angle to be free except in the upper nasal quadrant, where anterior synechias could be seen.

November 19.—Cyclodialysis was performed on the right eye, one third of the angle being opened.

November 20.—A thin membrane covered the surface of the vitreous, with a very small opening in its inferior aspect, through which vitreous had herniated.

November 24.—The patient was discharged from the hospital, with instructions to use 2 per cent pilocarpine nitrate three times a day in the right eye.

November 27.—Vitreous had herniated through the aforementioned opening sufficiently to be in contact with the cornea in one place below the pupillary border. Tension was 10 mm. in the right eye and 20 mm. in the left eye. Pilocarpine medication was stopped in the right eye and continued in the left eye.

December 4.—Examination revealed essentially no change. Vision was 20/30— in the right eye.

Feb. 28, 1946.—The vitreous had herniated farther into the anterior chamber of the right eye, so that it filled about three quarters of the anterior chamber and was in contact with the cornea over a considerable area, with two points of corneal edema. There was bulging of the iris at the 2 o'clock position. Tension was 26 mm. in the right eye, and vision was 20/40—. Operation was advised, but the patient requested postponement.

March 14.—With the Graefe knife, transfixion of the iris of the right eye was done in the 2 o'clock position. Considerable aqueous escaped, more than could have been present in the anterior chamber alone. The anterior chamber, which had previously been slightly shallower than would be expected in an aphakic eye, deepened in its inferior portion.

March 15.—The entire anterior chamber was deeper than previously. The vitreous was no longer in contact with the cornea. The patient was discharged, with instructions to use 1 per cent atropine twice a day. (Pilocarpine treatment was continued in the left eye.)

March 19.—There was some injection of the right eye. The vitreous had withdrawn further. Tension was 19 mm. in the right eye. The use of atropine was continued.

March 26.—The right eye was somewhat irritable, probably from atropine. The eye felt softer, but the tension was not taken because of the irritability. The vitreous was further retracted. Atropine medication was discontinued.

April 2.—The right eye was white and quiet. The vitreous was unchanged; it filled about one-half the anterior chamber in the pupillary area and was not in contact with the cornea. Tension was 18 mm. in the right eye.

April 16.—Tension was 17 mm. and vision 20/30 in the right eye.

May 27.—Tension was 13 mm. in the right eye. There was possibly a little more vitreous in the anterior chamber.

CASE 8.—L. S., a woman aged 65.

Sept. 25, 1942.—An immature cataract was present in each eye. Vision was 1/200 in the right eye and 4/200 in the left eye.

November 14.—Combined intracapsular cataract extraction was done on the right eye. Convalescence was uneventful. Final vision with glass was 20/20. This vision has been maintained to date.

November 21.—Extracapsular cataract extraction with iridectomy was done on the left eye. The convalescence was uneventful except that there was a pupillary membrane, partly capsular and partly inflammatory.

Jan. 30, 1943.—Discission was done on the left eye. A good opening was obtained. Subsequent vision with glass was 20/25.

Feb. 23, 1946.—The patient returned to the clinic with a history of severe pain and inflammation in the left eye, with loss of vision of six days' duration. The left eye was considerably injected; the cornea was steamy. The anterior chamber was somewhat shallow, particularly on the sides. It was deeper below. The pupil was slightly drawn up, and the coloboma of the iris was rather narrow, about 4 mm. Some of the capsule was present in the top of the coloboma and along the nasal border. The pupil was clear in the lower central portion, with vitreous herniating through it so as to reach almost to the cornea. Vision with glass was 15/70. Tension was 52 mm. A single puncture was made with a broad cataract knife in the periphery of the iris at the 4 o'clock position. The anterior chamber deepened immediately. A single dressing was applied, and the patient was allowed to return home.

February 25.—The left eye was almost white and quiet. The anterior chamber was deep. The vitreous had withdrawn from the anterior chamber, and its anterior surface was on the plane of the iris. Tension was 20 mm. No local treatment was given.

March 2.—The left eye was white and quiet. Tension was 23 mm.

March 11.—Tension was 14 mm. in the left eye.

March 25.—Vision with glass was 20/40—1 in the left eye. Tension was 15 mm. The anterior chamber was slightly shallow temporally. The vitreous was bulging slightly through the nasal third of the pupil.

April 22.—Tension was 16 mm. in the right eye and 15 mm. in the left eye. In the left eye, the vitreous was on the plane of the iris all over the pupil except for a very slight bulge on the nasal side. In several places there seemed to be a space between the iris and the vitreous.

CASE 9.—A. D., a man aged 68.

June 11, 1945.—On the patient's admission to the hospital, the right eye was aphakic, with iridectomy upward. The media were clear, and the fundus was normal. Vision with glass was 20/30 in this eye. The anterior chamber of the left eye was normal and the pupil round and active. Vision was 20/200 in this eye.

June 12.—Intracapsular cataract extraction, with a small peripheral iridectomy, was done on the left eye. There were no complications.

June 14.—The anterior chamber was good. The pupil was round and central. There was moderate redness.

June 19.—The patient was discharged. Atropine was prescribed. Vision with a + 12.00 D. sphere and a pinhole disk was 20/30—.

June 25.—The left eye was moderately injected. The cornea was clear. Sutures were removed.

July 16.—The anterior chamber of the left eye was rather shallow. There was moderate injection.

July 30.—The left eye was moderately injected. The anterior chamber was shallow. The pupil measured about 2.5 mm. The hyaloid membrane was on the plane of the iris. Tension was 48 mm. Four per cent pilocarpine nitrate was prescribed.

July 30.—Tension was 48 mm. in the left eye. Vision with a + 12.00 D. lens and a pinhole disk was 20/30. Vitreous was bulging somewhat through the pupil, but the hyaloid membrane was intact. The iridectomy opening seemed closed with a membrane. The anterior chamber was very shallow. The patient was admitted to the hospital.

August 3.—Cyclodialysis was done on the left eye, between 6 and 9 o'clock.

August 4.—Tension was 36 mm. in the left eye. A 4 per cent solution of pilocarpine nitrate and physostigmine ointment were prescribed.

August 8.—Tension was 36 mm. in the left eye.

August 13.—The patient was discharged. Tension was 28 mm. in the left eye.

August 20.—The patient was admitted to the hospital. Vision in the left eye with a + 12.00 D. lens and a pinhole disk was 20/30—. Tension was 48 mm. The anterior chamber was very shallow.

August 25.—Trephination was done on the left eye, with a small iridectomy. Vitreous was lost at operation.

August 28.—The anterior chamber was rather shallow. Tension was 36 mm. Four per cent pilocarpine nitrate and 1 per cent epinephrine hydrochloride were given, and daily massage of the eye was prescribed.

September 5.—Tension was 35 mm. in the left eye.

September 11.—Tension was 45 mm. in the left eye.

September 11.—Cyclodiathermy of the lower half of the circumference of the eye and posterior sclerotomy in the lower outer quadrant were performed.

September 12.—The anterior chamber had become a little deeper. The eye was soft after massage.

September 19.—Tension was 18 mm. in the left eye. The anterior chamber was a little deeper than before but was still rather shallow. The pupil was small. The vitreous showed a great deal of opacity. Vision equaled accurate light pro-

jection. The patient was discharged from the hospital. Four per cent pilocarpine nitrate was prescribed.

September 24.—The left eye was still rather red. The pupil measured 2 mm. and was fixed to light. The anterior chamber was very shallow. There was some blood in the pupil. Tension was normal to palpation. Atropine was prescribed.

October 1.—The anterior chamber was very shallow. The pupil was small. A posterior synechia was present. Tension was 36 mm. The patient was admitted to the hospital.

October 2.—A single small puncture was made in the lower outer quadrant of the iris.

October 4.—Tension was normal to palpation. The tiny puncture in the iris seemed to be filled with blood and fibrin, and iris was adherent to the back of the cornea here. The anterior chamber was somewhat deeper than before.

October 8.—Tension was 45 mm. in the left eye.

October 9.—A small cyclodialysis was done in the upper nasal quadrant, and a broad basal iridectomy was done through the cyclodialysis wound. No vitreous was lost. Moderate bleeding occurred into the anterior chamber.

October 10.—The anterior chamber was deep.

October 17.—The anterior chamber was deep. Nearly all the blood had gone from the anterior chamber. The eye was "soft."

October 22.—The eye was "soft." Vitreous was bulging somewhat into the anterior chamber. Atropine was prescribed.

November 5.—The left eye was "soft." Atropine medication was continued.

November 19.—Tension was 10 mm. in the left eye. Atropine medication was discontinued. The vitreous was clearing.

December 3.—The left eye was "soft."

December 24.—Tension was 18 mm. in the left eye, and the eye was still moderately injected.

Jan. 21, 1946.—Examination of the left eye showed a few cells in the aqueous, a little blood around the margin of the coloboma of the iris and many adhesions between the iris and the hyaloid membrane, forming a complete synechia except in the coloboma. Many coarse opacities were seen in the vitreous. The eye was nearly white and quiet. The disk was slightly pale. No pathologic cupping was present. Tension was 14 mm. Vision with glass was 20/50.

February 4.—Tension was 19 mm. in the right eye and 15 mm. in the left eye. Both eyes were white and quiet. There was no pathologic cupping, but the left disk was slightly paler than the right. The anterior chambers were deep. A few large opacities were present in the vitreous of the left eye. Only a little blood clot remained around the border of the coloboma in the left eye.

March 4.—Tension was 18 mm. in the right eye and 14 mm. in the left eye. In the left eye, the posterior synechia between the iris and the hyaloid membrane was complete everywhere except in the operative coloboma.

April 1.—Tension was 17 mm. in the right eye and 14 mm. in the left eye. Examination showed no change.

CASE 10.—L. V., a man aged 50.

Feb. 16, 1945.—Three months prior to admission the patient had been struck in the left eye with a blunt piece of metal. He was said to have had a severe

intraocular hemorrhage with glaucoma, necessitating irrigation of the anterior chamber to remove the blood.

Right Eye: Vision was 20/20, and the eye was normal.

Left Eye: The eye was slightly injected. There was a scar about 6 mm. long in the cornea near the limbus at 12 o'clock, with iris adherent to it. The pupil was slightly drawn up, about 6 mm. horizontally and 8 mm. vertically. It reacted to light. The lens was in position above and tilted slightly backward below, with vitreous presenting in the space between the iris and the lens. The lens showed considerable opacity. The aqueous showed a few cells, and the beam of the slit lamp was slightly visible. Tension was normal. Vision was 6/200. Atropine was prescribed.

During the next few months the eye gradually became quiet and the lens completely opaque.

September 7.—Operation was performed on the left eye. Two corneal sutures were used. An upward incision was made with a Graefe knife, and the anterior synechia was cut through in making the section, producing a fairly large coloboma of the iris upward and leaving some iris attached to the cornea. Normal vitreous presented immediately, and the lens was delivered in capsule with a wire loop. There was considerable loss of vitreous. A large bubble of air was put into the anterior chamber. The chamber formed the day after operation. The patient was discharged on the seventh day after operation, and atropine was prescribed. There was considerable postoperative inflammation, and the anterior chamber remained very shallow.

October 5.—The eye was almost white. The anterior chamber was extremely shallow. No free vitreous was present in the chamber. What appeared to be a hyaloid membrane stretched across the pupil. Tension was 25 mm. Atropine medication was discontinued.

October 15.—The anterior chamber was extremely shallow. Hyaloid membrane was in contact with the cornea in the upper central zone. The pupillary border was everywhere in close contact with the hyaloid membrane, even as far up into the coloboma of the iris as could be seen. Many fine synechias were present between the iris and the hyaloid membrane. The pupil was in the same state of dilatation as at the first examination and did not react to light. Tension was 38 mm. Four per cent pilocarpine nitrate was ordered.

October 18.—Tension was 40 mm. The eye was otherwise unchanged. The patient was admitted to the hospital for operation. A conjunctival incision was made in the lower outer quadrant and a flap dissected up toward the limbus. A scratch incision about 6 mm. long was made through the sclera parallel to, and about 5 mm. back of, the limbus. A spatula was introduced and a small cyclodialysis opening made. Iris forceps were introduced into the anterior chamber; the iris was drawn out through the scleral wound, and a broad iridectomy was done. No vitreous was lost. Considerable bleeding occurred into the anterior chamber. The next day the anterior chamber was deep, as in the usual aphakic eye, and tension was normal. Considerable blood was present in the chamber. The patient was discharged on the sixth postoperative day. At discharge gross blood was gone from the anterior chamber. The chamber everywhere was deep except where the strip of iris between the upward coloboma and the recent iridectomy was in contact with the back of the cornea.

October 31.—Tension was 27 mm. The patient was given physostigmine and epinephrine and told to massage the eye.

November 9.—Tension was 17 mm.

November 16.—Tension was 19 mm. Four per cent pilocarpine nitrate and 1 per cent epinephrine hydrochloride were prescribed.

Subsequent Course.—The patient was seen at weekly or semiweekly intervals until January 18. At these visits, except for two readings of 30 mm., tension remained 25 mm. or less. The anterior chamber remained deep; the eye became quiet. There was moderate opacity of the vitreous, and no cupping of the disk.

It is probable that the angle is sufficiently blocked by anterior synechia from the long-continued extreme shallowness of the chamber so that further operation will ultimately be necessary permanently to control the tension.

CASE 11.—L. C., a man aged 66.

March 11, 1945.—Immature cataract was present in both eyes. Vision was 20/70 in the right eye and 20/50 — in the left eye.

June 17.—An uneventful intracapsular cataract extraction with complete iridectomy upward was performed on the right eye. Two corneoscleral sutures were used. The anterior chamber formed on the day following operation. The patient was discharged on the ninth postoperative day and given atropine.

August 6.—The patient did not report back to the clinic until this date. At this visit the eye showed considerable injection and slight striate keratitis. There was a good anterior chamber. There were numerous opacities in the vitreous, so that details of the fundus were not made out. Examination with the slit lamp was not carried out. The patient complained of some pain in the eye. There was no note of tension.

August 9.—The patient returned with severe pain in the right eye. The anterior chamber was extremely shallow, especially temporally, where the iris seemed to be in contact with the cornea. The cornea showed epithelial edema. Many cells were noted in the aqueous and many opacities in the vitreous. Tension was 30 mm. Two per cent pilocarpine nitrate was prescribed.

August 12.—The patient was admitted to the hospital, with marked injection of the right eye, a steamy cornea and an extremely shallow anterior chamber, which seemed to be flat temporally. He was suffering from severe pain. Tension was 48 mm. Paracentesis was done through the cornea in the 10 o'clock position with a Graefe knife. When the knife was withdrawn, the iris prolapsed through the wound. When an attempt was made to replace it, a hole was inadvertently made in the iris, and a gush of watery fluid escaped through the wound. The iris was then easily reposited. The following morning the eye was described as "mushy soft." The anterior chamber was deep. Atropine was prescribed and its use continued during the stay in the hospital. Intravenous injection of typhoid vaccine was given on two occasions. The eye slowly quieted down, and the patient was discharged on the eighteenth postoperative day. Tension remained normal and the anterior chamber deep. Atropine medication was continued at home.

September 13.—There was slight ciliary injection. The anterior chamber was deep. There was a thin pupillary membrane, and numerous cells were noted in the aqueous. Atropine medication was continued.

October 8.—The right eye was practically white and quiet. A rare cell was present in the aqueous. Use of atropine was discontinued.

November 19.—The right eye was white and quiet. A thin pupillary membrane was present. Tension was 20 mm.

Feb. 18, 1946.—Vision with a + 10.00 D. lens and a pinhole disk was 20/30—1 in the right eye. Tension was 19 mm.

February 27.—Refraction of the right eye was done, and vision with glass was 20/30.

CASE 12.—P. F., a man aged 59.

Jan. 31, 1944.—An immature posterior cortical cataract was present in both eyes. Vision was 2/200 in the right eye and 20/40 in the left eye.

March 7.—Intracapsular cataract extraction with peripheral iridectomy was done on the right eye. There was no loss of vitreous or other complication. The anterior chamber was formed on the first postoperative day, and convalescence in the hospital was uneventful. The patient was discharged on the ninth postoperative day. Atropine was prescribed.

March 27.—The eye was moderately red.

April 17.—There was a history of pain for five days. The right eye was moderately red. The pupil measured 3 mm. The cornea was edematous. The anterior chamber was shallow. There were a few cells in the aqueous. Tension was 48 mm. A Reese incision was made into the anterior chamber.

April 20.—Tension was 49 mm. in the right eye. Another Reese incision was made.

April 25.—Posterior sclerotomy was done in the upper nasal quadrant.

April 29.—Tension in the right eye was normal to palpation. The anterior chamber had become slightly deeper.

May 1.—Tension was 42 mm. in the right eye. The eye was considerably injected. The pupil measured 3 to 4 mm. and was fixed to light. The anterior chamber was moderately shallow. Vision was 3/200.

May 8.—The anterior chamber was moderately deep. There was no pupillary reaction to light. A few cells were present in the anterior chamber. "Vitreous was also present." Tension was 36 mm.

May 16.—Cyclodialysis was done in the lower outer quadrant.

May 19.—Tension was 65 mm.

May 20.—A trephine opening was made in the upper inner quadrant, with a small iridotomy. There was no loss of vitreous.

May 22.—Tension was 22 mm.

May 24.—Tension was 40 mm.

May 25.—Tension was 36 mm. With massage and use of 1 per cent epinephrine hydrochloride, tension gradually fell to normal, and the patient was discharged June 3.

June 12.—Tension was 36 mm. Use of 4 per cent pilocarpine nitrate was continued.

June 26.—Tension was 30 mm. A small infiltrated ulcer was noted in the lower portion of the cornea, with a hypopyon measuring 3 mm. The pupil was here described as being drawn somewhat upward and nasally, so as to give the appearance of a complete coloboma. The anterior chamber was deep. With intravenous injections of typhoid vaccine and local administration of penicillin, the ulcer gradually healed. Tension became higher, however, and on June 30 a paracentesis of the anterior chamber was done.

July 4.—Tension was 40 mm.

July 5.—Cyclodiathermy was done on the temporal side.

July 11.—The patient was discharged from the hospital. Tension was normal to palpation. Determinations of tension were made as follows:

Date	Tension, Mm.
July 17	17
July 24	22
July 31	22
August 14	"Eye still red"
September 11	45

September 12.—Cyclodiathermy was done at the 7 to 10 o'clock position.

September 30.—The patient was discharged from the hospital. The right eye was soft to palpation. Determinations of tension were made as follows:

Date	Tension
October 16	Very soft
November 6	Very soft
December 4	Very soft

Jan. 8, 1945.—Tension was 22 mm. in the right eye, and vision with glass was 6/200. During the next several months the right eye remained quiet, and tension was usually 22 mm. or less.

December 10.—Tension was 42 mm., and vision was 3/200.

CASE 13.—J. T. H., a man aged 87.

Dec. 24, 1945.—The patient was admitted to the hospital with a history of pain and blurred vision in the right eye for forty-eight hours. The right eye was considerably injected, the cornea steamy and the anterior chamber deep; the pupil was about 2 mm. and was fixed to light. The iris was tremulous. There was a hypermature cataract, which was subluxated backward and slightly nasally, and vitreous was bulging into the nasal part of the pupil. There was marked exfoliation of the lens capsule. Tension was 42 mm. Vision equaled accurate light projection. The left eye was white and quiet; the anterior chamber was deep, and the pupil was about 2.5 mm. and reacted to light. There was an immature cataract. Vision was 20/100. Tension was 16 mm.

During the day of admission the right eye was given intensive treatment with 4 per cent pilocarpine nitrate and 0.5 per cent physostigmine salicylate. At 8:30 p. m. tension in the right eye was 32 mm. While the patient was in the hospital, 4 per cent pilocarpine nitrate and 1 per cent epinephrine hydrochloride were used in the right eye four times a day. Tension remained normal, and he was discharged December 26, with instructions to follow the same treatment.

December 31.—Tension was 18 mm.

Jan. 5, 1946.—Tension was 19 mm.

January 7.—The patient was readmitted to the hospital with a history of severe pain in the right eye for eighteen hours. The eye was intensely injected; the cornea was steamy; the anterior chamber was almost flat, and the pupil measured 3 mm. and was fixed to light. On account of the steamy cornea, it was difficult to tell whether there was vitreous in the pupil. The lens appeared to be 2 or 3 mm. behind the plane of the iris. Tension was 46 mm.

An operation was done, consisting of a double transfixion of the iris in the upper portion with a Graefe knife. There was considerable escape of aqueous, and the eye became soft.

January 8.—The anterior chamber was deep and tension was normal. No treatment was given.

January 10.—Tension was 17 mm. The patient was discharged from the hospital without treatment.

January 14.—The cornea was clear, the anterior chamber deep and the vitreous bulging through the pupil on the nasal side. The lens was tilted backward nasally. The two holes in the iris were open, but there was an anterior synechia at the site of the nasal hole.

January 28.—Tension was 30 mm. The right eye was moderately injected. The anterior chamber was deep except for an anterior synechia at the site of the nasal one of the two iris punctures. Both holes were patent. The chamber was also slightly shallow at the periphery inferotemporally. The pupil was about 3 mm.; vitreous filled the pupil but did not project into the anterior chamber.

It was felt at this examination that the slightly increased tension was due to shallowing of the anterior chamber peripherally in the lower temporal quadrant from the backward tilting of the lens, together with the shallowing incident on the formation of the anterior synechia at the site of the temporal puncture and the presence of exfoliation of the lens capsule. In view of this, it was felt that the glaucoma should now be favorably influenced by a miotic. The patient was given 2 per cent pilocarpine nitrate to use four times a day.

February 11.—Tension was 26 mm. in the right eye and 17 mm. in the left eye. Examination revealed no change.

February 25.—Tension was 28 mm. in the right eye and 15 mm. in the left eye. Pilocarpine nitrate, 4 per cent, was given four times a day in the right eye.

March 25.—Tension was 34 mm. in the right eye and 15 mm. in the left eye. The right eye was essentially unchanged in appearance; vitreous still filled the pupil. The anterior chamber was everywhere deep except in the lower outer quadrant. The pupil was 3 mm. and fixed. Exfoliation was marked. The patient had neglected to use drops. They were again ordered.

April 22.—Tension was 24 mm. in the right eye and 20 mm. in the left eye. With the slit lamp, a little vitreous was seen protruding slightly into the anterior chamber through the lower half of the pupil. Medication with 4 per cent pilocarpine nitrate was continued.

CASE 14.—J. M., a man aged 75.

Jan. 29, 1945.—Right Eye: The cornea was clear; the anterior chamber was slightly shallow; the pupil was round and active; an immature cataract was present, with marked exfoliation of the lens capsule, and tension was normal to palpation. Vision was 11/200.

Left Eye: The eye was slightly convergent; the anterior chamber was deep; the lens was completely dislocated posteriorly and lying down behind the iris below; the fundus was normal. Tension was normal to palpation. Vision with a cataract glass was 20/30. Glasses were prescribed.

December 31.—The patient complained of blurred vision in the left eye. The disk was slightly pale, but no pathologic cupping was noted. Vision was 20/70 with glass. Tension was 25 mm. Two per cent pilocarpine nitrate was prescribed.

Jan. 7, 1946.—Vitreous was noted in the anterior chamber. Tension was 46 mm. Admission to the hospital was advised.

January 14.—The patient was admitted to the hospital. The right eye was unchanged; tension was 17 mm.

Left Eye: The anterior chamber was almost normal, possibly slightly shallow, and of about the same depth as that of the right eye. Examination with the slit lamp showed vitreous in the pupil and nearly filling the anterior chamber.

It was thought to be normal, since it was tremulous. Tension was 57 mm. Vision with glass was 20/200.

January 15.—Operation was performed on the left eye. A double transfixion of the iris was done in the upper portion with a Graefe knife, and the eye immediately became soft. A large bubble of air was introduced into the anterior chamber; since this made the eye hard, it was evacuated. When the air was evacuated, it was followed by a bead of vitreous, which was pulled out and excised. Atropine was prescribed.

January 16.—The anterior chamber of the left eye was rather deep; tension was normal.

January 17.—Tension was 18 mm. in each eye.

January 18.—Tension was 17 mm. in each eye.

January 19.—The patient was discharged. The anterior chamber in the left eye was rather deep and was nearly filled with vitreous. The pupil was about 3.5 mm. Tension was 14 mm.

January 22.—Tension was 16 mm. in the right eye. The left eye still showed moderate injection. The pupil was 3 to 4 mm. Tension was 23 mm. Atropine medication was continued.

January 28.—Tension was 19 mm. in the right eye and 30 mm. in the left eye.

Left Eye: A strand of vitreous extended from the pupillary area slightly upward and outward to the corneal incision. The lower part and the nasal side of the anterior chamber was free of vitreous. Atropine was discontinued, and 2 per cent pilocarpine nitrate was substituted.

February 18.—Tension was 38 mm. in the right eye and 17 mm. in the left eye.

Left Eye: Vitreous still protruded through the pupil, partially blocking it. Some vitreous presented in one of the transfixion holes in the iris.

Right Eye: The eye was hard to palpation, with much exfoliation, steamy cornea, shallow anterior chamber, moderate conjunctival injection and mature cataract. With treatment with 4 per cent pilocarpine nitrate, the tension in the right eye dropped, varying from 15 to 26 mm. until February 26.

February 26.—An intracapsular cataract extraction with basal iridectomy was done on the right eye. There was no loss of vitreous, but the anterior chamber formed while the patient was still on the table. Postoperatively there was pronounced iridocyclitis with membrane formation; the patient was treated with atropine. He was discharged on March 9.

March 25.—Right Eye: There was slight ciliary injection; the eye was soft; there were a pupillary membrane and many opacities in the vitreous. The patient was using atropine.

Left Eye: Tension was 30 mm.; the patient was using pilocarpine. The same amount of vitreous was present in the anterior chamber.

April 8.—Right Eye: Tension was 14 mm. The sutures were removed. There was still a moderate mixed injection. The anterior chamber was deep. The pupil was drawn up. There was a fairly dense pupillary membrane, with a red reflex. Some folds were present in the cornea; there were no cells in the anterior chamber.

Left Eye: Tension was 27 mm. The eye was white and quiet. A strand of vitreous extended from the pupil to the lower end of the corneal incision. The anterior chamber was deep. The disk was nearly white.

Treatment: The right eye was treated with heat four times a day; the left eye was given pilocarpine nitrate, 4 per cent, four times a day.

April 22.—Tension was 34 mm. in the right eye and 20 mm. in the left eye. Examination showed no change in the left eye. Right Eye: The anterior chamber was shallow, especially above; a vascularized deep corneal opacity was present up and temporally from 8 to 1 o'clock, extending 2 mm. into the cornea. A dense pupillary membrane was present. The remains of a few small keratic precipitates were seen on the lower half of the cornea. There was slight epithelial edema of the upper third of the cornea. There were no cells in the anterior chamber and no aqueous flare. An emergency admission was advised but was refused by the patient.

April 29.—The patient was admitted to the hospital. Right Eye: Vision was limited to perception of hand movements. The anterior chamber was shallow; there were no cells, but many old pigmented keratic precipitates were present. The coloboma of the iris was filled with dense membrane. The fundus was not seen. Tension was 30 mm.

Left Eye: Examination showed no change. Tension was 21 mm. Vision was limited to counting of fingers at 4 feet (120 cm.). The disk showed shallow cupping.

April 30.—Operation was performed on the right eye. A double transfixion of the iris was done below. There was a little bleeding of the iris, and the anterior chamber deepened slightly.

May 2.—The patient was discharged. Vision with glass was 1/200 in the right eye and 20/100 in the left eye.

May 6.—Tension was 14 mm. in the right eye and 25 mm. in the left eye. The patient had not been using drops in the left eye. Medication was started again with 4 per cent pilocarpine nitrate, given four times a day.

Right Eye: The anterior chamber was deep. The transfixion holes were barely visible.

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ABSTRACT OF DISCUSSION

Dr. John H. Dunnington, New York: This timely paper focuses attention on some of the factors responsible for glaucoma in the aphakic eye. By so doing, the authors have made a noteworthy contribution and, consciously or unconsciously, have entered a plea for a more detailed preoperative study of each patient. They recognize that individualization in the treatment of these patients is essential. Such a clearcut analysis of the causative factors and the rationale for different methods of relief should make all ophthalmologists conscious of the futility of adopting one operative procedure as a routine measure in all cases of this condition.

In cases of type 1 the authors point out that the communication between the anterior and the posterior chamber may become blocked by a membrane, and they cite 3 cases in which this condition resulted in increased intraocular tension, relief being obtained by a discission in 2 of them. They caution against the use of this operative procedure in the presence of normal vitreous—an opinion in which I heartily concur, for in my experience the cutting of a secondary membrane in the presence of an increased intraocular tension usually leads to aggravation of the preexisting glaucoma. Considering the difficulty of determining the

exact status of the vitreous behind a dense membrane, the establishment of a communication between the anterior and the posterior chamber as an initial step is a safer operative procedure.

Herniation of the vitreous through the pupillary area does not of itself produce glaucoma. As pointed out by the authors, it occurs only when the communication between the anterior and the posterior chamber is blocked, a condition not infrequently seen after a discission. Such blocking adhesions between the iris and the prolapsed vitreous occur more readily in the presence of a postoperative iridocyclitis, the frequency of which is in direct proportion to the amount of operative trauma. For this reason, I have long contended that a discission is not a minor surgical procedure, but one that should be done with the greatest care and with minimal disturbance of the vitreous. To relegate this operation to the novice is to increase the frequency of postoperative glaucoma. A careful preoperative study of the membrane will disclose how a satisfactory opening can be made in it with the smallest amount of trauma. The frequency of this type of glaucoma can be reduced materially by remembering that a small opening in a secondary membrane, if placed in the proper position, gives the same visual result as a wide one. Above all, unnecessary disturbance of the vitreous should be avoided.

The occurrence of glaucoma from herniation forward of the vitreous into the anterior chamber has occurred in several of my cases of intracapsular extraction, both with and without complete iridectomy. In most of them convalescence was complicated by a low grade cyclitis, which was followed in a short time by a rise in the intraocular tension. In these cases I have always recommended a cyclodialysis with iridectomy, as described by Wheeler in 1936, as the operation of choice. In view of the authors' findings, I believe that either a transfixion of the iris or an iridectomy should be considered, for I have long observed that a cyclodialysis, unless combined with an iridectomy, is ineffective in many cases of glaucoma with aphakia.

Herniation of the vitreous forward, blocking the communication between the anterior and the posterior chamber, may be the cause of the rise in tension that follows a delayed restoration of the anterior chamber. In 1 case of this type a restoration of normal intraocular tension followed the freeing of an adhesion between the anterior face of the prolapsed vitreous and the posterior surface of the cornea, whereas a previous cyclodialysis had failed to reduce the tension.

In some instances the increased tension resulting from a herniation of vitreous through the round pupil can be relieved by medical means. Restoration of the mobility of the iris by first dilating and then contracting the pupil was sufficient to control the tension in 2 of my cases. Let me emphasize the importance of detailed examination with the slit lamp as a part of the postoperative follow-up in cases of cataract extraction, particularly those with a round pupil. Such a herniation of the vitreous can be easily seen, and if it is associated with an immobile iris, beware! Similarly, the early detection of adhesions between the iris and the nonprolapsed (anterior) face of the vitreous should enable one to prevent the development of glaucoma from that cause. The failure of the authors to find any reference in the literature to cases of this sort is but proof that sufficient attention has not been given to the mobility of the iris.

Their case reports clearly demonstrate that a blockage of the pupil by vitreous may be responsible for glaucoma in some patients with a dislocated lens, and their method of treatment warrants serious consideration. My experience with cases of this type has been confined to the removal of the lens combined with a complete iridectomy; but, thanks to the authors, henceforth I shall always bear in mind that vitreous block is a factor to be reckoned with.

Dr. Paul A. Chandler, Boston: Of all the types of glaucoma I have treated, this affords the greatest satisfaction to the surgeon. If a proper communication is reestablished between the chambers, so far as our present experience goes with the 14 cases discussed in this paper and with 3 or 4 others which are not included here, the results are uniformly successful. In case 8, in the second group, in which glaucoma came on after a needling, a simple puncture of the iris was done. A single dressing was applied; the patient was sent directly home and made a rapid and uneventful recovery.

A large herniation of the vitreous into the anterior chamber may occur without a pupillary block, and it is rare that the entire pupil is blocked with vitreous if a complete iridectomy has been done. It is known that there is a continuous exchange of fluid between the vitreous and the aqueous all the time; probably in these cases there is a little leakage of aqueous between the pupillary border and the herniating vitreous, but it is apparently not sufficient to make free communication between the chambers. The most dramatic proof to my mind that the vitreous can block the pupil is that when a communication is reestablished between the chambers one can see the vitreous withdraw from the anterior chamber.

Dr. Dunnington referred to the use of drops. If the pupil is blocked with vitreous, the drug of choice is atropine, instead of pilocarpine. Pilocarpine makes the condition worse, as was proved in several of our cases, because it makes the pupil grip the herniating vitreous all the more firmly and increases the degree of pupillary block.

MINOR ANOMALIES IN POSITION OF THE EYELASHES

FRITZ J. BLOCH, M.D.
NEW YORK

CILIUM INCARNATUM EXTERNUM

THE SUBCUTANEOUS growth of an eyelash is infrequently reported. Makrocki [1] described the case of a woman aged 22 with two small nodules in each upper lid which proved to be eyelashes 3 to 4 mm. in length. Except for unusually heavy pigmentation, they were normal. Herzog [2] described the case of a woman aged 24 with a wart the size of a pinhead on the outer margin of the left upper lid. On the inner circumference of the base of the wart a cilium was bent backward and grew along the margin of the lid, completely surrounded with epithelium. Slitting of the epithelium permitted the liberation of a normal eyelash to its normal position. Cherno [3] mentioned as *Bedeutungsvolles Kuriosum* a case in which a normally formed, extremely thick eyelash 8 mm. long was found under the skin of the lid. Schreiber [4] observed a 17 year old girl with a comblike vertical elevation of the skin immediately above the margin of the lid, completely surrounding an eyelash. Jess [5] described the case of a 19 year old patient with a longitudinal swelling of the skin of the upper lid through which a cilium could be seen as a black stripe growing upward.

I observed a similar case, that of a man aged 39 who consulted me because he had difficulty in removing a blackhead from his left upper lid. Two millimeters nasal to the midline the left upper lid showed a longitudinal elevation of darker color than the surrounding skin. Examination with the slit lamp showed a cilium completely surrounded with epithelium. After a small incision with a Graefe knife near the margin of the lid, the cilium, 7 mm. long, could easily be extracted with forceps. Apparently normal in all respects, it joined the patient's normal row of lashes. There was no other pathologic condition of the eyes (figure, *A*).

From the service of Dr. E. B. Gresser, Department of Ophthalmology, Beth Israel Hospital.

1. Makrocki, F.: Ein Fall von pervers gewachsenen subcutanen Cilien, Zentralbl. f. prakt. Augenh. **7**:129,1883.

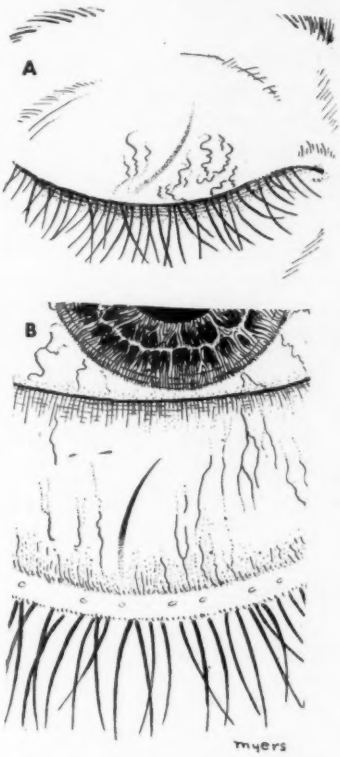
2. Herzog, H.: Pathologie der Cilien, Ztschr. f. Augenh. **12**:256, 1904.

3. Cherno, F.: Ueber die unter dem Namen Blepharitis ciliaris bekannten Erkrankungen des Lidrandes, Ztschr. f. Augenh. **18**:1, 1907.

4. Schreiber, L.: Die Krankheiten der Augenlider, Berlin, Julius Springer, 1924, p. 380.

5. Jess, A.: Cilia incarnata totalis, Klin. Monatsbl. f. Augenh. **83**:47, 1929.

Comment.—This anomaly may have little practical importance, but the apparent rarity and the fact that no case has been published in the Anglo-American literature may justify its report. For the explanation of the condition it is not necessary to go into a far-fetched embryologic discussion, as Makrocki [1] did. The sprouting eyelash encounters resistance, such as a wart or a large infarct of a meibomian gland, in



Artist's sketches of (*A*) cilium incarnatum externum and (*B*) cilium incarnatum internum.

its growth. On being bent upward, it follows the line of least resistance, finding its way under the skin of the lid.

CILIUM INCARNATUM INTERNUM

Cilium incarnatum internum is a rarer condition than the preceding one. In this form an aberrant cilium finds its way through the conjunctiva tarsi and is much more unpleasant to the patient. Eisner [6] observed a 50 year old farmer who complained of a stinging pain in his right

6. Eisner, F.: Beitrag zur Anomalie des Zilienwachstums, Klin. Monatsbl. f. Augenh. **85**:810, 1930.

eye for a few days. There was an erosion of the lower part of the cornea, caused by the tip of an eyelash which perforated the conjunctiva 1.5 mm. below the margin of the lid. The cilium was easily removed, but it kept on growing back in this anomalous position and had to be removed again twice in the course of three years. Hirose and Hirayama [7] reported a similar case, that of a 17 year old girl, in which a black hair was directed upward in the tarsal conjunctiva of the upper lid. The hair was found to be an eyelash.

I observed a similar case in a man aged 50 whose eyes had been normal at a previous examination. He complained that he had felt a scratching sensation in his right eye for the previous two weeks. The eye became increasingly red. Examination showed moderate injection of the lower part of the conjunctiva bulbi. The corneal surface was not as brilliant as normal but did not stain. After removal of mucus from the conjunctiva tarsi, examination with the slit lamp showed a hair 2 mm. below the margin of the lid in the midline. It was visible about 1.5 mm. below the conjunctiva, and an end of the same size was moving freely in the cul-de-sac. A light pull with the forceps extracted the hair, which proved to be an eyelash, rather thin, little pigmented and slightly curled at its end. Two days later the patient presented himself as cured (figure, *B*).

Comment.—The ingrowth of an eyelash and its appearance in the conjunctiva can be explained on a strictly mechanical basis, in the same way as cilium incarnatum externum. Before perforating the lid margin, the cilium, meeting an obstacle, is bent backward and enters by chance the lumen of a meibomian gland. It grows backward along the gland until it perforates the tarsus and the conjunctiva. However, there is a possibility that one is dealing with a congenital false hair anlage in which the root of the cilium faces the margin of the lid. Such a condition is known as *cilium inversum,* and a histologic specimen has been reproduced by Schreiber.[8]

SUMMARY

Two cases of anomalous upward growth of an eyelash are described. In the first case the cilium found its way into the outer skin of the lid; in the second case it perforated the conjunctiva, and its tip moved freely in the cul-de-sac.

114 East Fifty-Fourth Street.

7. Hirose, K., and Hirayama, M.: An Anomaly of the Eyelashes, Acta Jap. Ophth. Soc. **3**:108, 1932; abstracted, Arch. Ophth. **10**:681 (Nov.) 1933.

8. Schreiber,[4] p. 381.

Clinical Notes

LATENT NYSTAGMUS FOLLOWING HEAD INJURY

GEMINIANO de OCAMPO, M.D., MANILA, PHILIPPINES

IN ORDER to find a satisfactory explanation for the signs and symptoms observed in a case of latent nystagmus, the literature on the subject has been surveyed and has been found not only wanting but with many contradictions. Kestenbaum [1] stated that the mechanism and genesis of latent nystagmus are still controversial and indefinite, while Duke-Elder [2] called the explanations of various authors quite hypothetical. According to Esters,[3] the cause of the phenomenon is unknown. The purpose of this report is not to offer another theory to the many already advanced but, rather, to attempt to integrate and coordinate them for a better understanding of this interesting phenomenon, exemplified by a case following head injury.

There seems to be no doubt that latent nystagmus is a disturbance or perversion of the normal mechanism of extraocular muscle tonus, fixation and conjugate ocular movements. It is equally evident that, while many of their phases have been well worked out, there remain some controversial points difficult of experimental proof. There is definite evidence that the movements in latent nystagmus are conjugate and bilateral, and it appears in the light of present concepts that they must primarily be controlled from a coordinating center, wherever and whatever that is. While a supranuclear center for normal conjugate movements is generally accepted, such a center for latent nystagmus has been called by various names, such as "main ocular muscle sender," by Ohm [4]; "association centers," by Fuchs [5]; "lower centers," by Posner [6]; "coordinating cen-

From the Department of Ophthalmology and Otolaryngology, University of the Philippines College of Medicine.

1. Kestenbaum, A.: Clinical Methods of Neuro-Ophthalmologic Examination, New York, Grune & Stratton, Inc., 1946, p. 230.

2. Duke-Elder, W. S.: Text-Book of Ophthalmology, London, Henry Kimpton, 1942, vol. 1, p. 638.

3. Esters, cited by Duke-Elder.[2]

4. Ohm, cited by Kestenbaum, A.: Physiological and Clinical Aspects of Nystagmus, in Ridley, F., and Sorsby, A.: Modern Trends of Ophthalmology, London, Butterworth & Co., Ltd., 1940, pp. 372-381.

5. Fuchs, E.: Textbook of Ophthalmology, ed. 5, edited by A. Duane, Philadelphia, J. B. Lippincott Company, 1907, pp. 800-801.

6. Posner, A.: A Contribution to the Theory of Binocular Vision Supported by Three Cases of Latent Nystagmus, Am. J. Ophth. **28**:392-396 (April) 1945.

ters," by Fromaget [7]; "supranuclear center," by Verhage,[8] and "center of rhythmic coordination," by Rea.[9] Bielschowsky's [10] "anomalous innervation centers" is probably related to it. Whether this center is single, bilateral or multiple, the terms refer to the same thing physiologically. Wherever it is anatomically, whether in Deiters' nucleus, according to Ohm [4]; in the quadrigeminal bodies, according to Whitnall,[11] or elsewhere in the mesencephalon, it must be connected directly or indirectly with the common motor pathways of the third, fourth and sixth nerves to the ocular muscles and with the many reflex peripheral sensory pathways, such as those from the labyrinth and the pathways mediating propioceptive impulses from the muscles of the neck, light impulses from the retina and accommodation, convergence and fusion impulses, as well as the involuntary central impulses or innervations from the occipital lobes and the cerebellum and the voluntary innervation from the frontal lobes.

Many believe with Duane [12] that the act of fixation is influenced simultaneously under normal conditions by the left and the right frontal as well as the occipital cerebral motor centers. When the eyes are in the primary position, the right and the left centers exert equal innervations, but when looking to the side, as to the right, the innervations, whether voluntary or reflex, although bilateral, are mainly from the left, or opposite, side, and vice versa. These central innervations are thought of as inhibitory and steadying (Posner [6]), or as actually stimulating to the impulses from the lower centers.

What is obscure, however, is whether these supranuclear lower centers are not generators, or the source of innervations of their own (Posner,[6] Ohm [4]) as well as coordinators. If they are not generators, then the extraocular muscle tone present after cerebral influence is removed must be from constant tonic innervations from the cerebellum or from propioceptive impulses from the extraocular muscles through the third, fourth and sixth nerves (Sherrington [13]).

It is evident from this discussion that, while much depends on the congenital or acquired integrity or capacity of the coordinating mecha-

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7. Fromaget, cited by Duke-Elder.[2]

8. Verhage, J. W.: Eine klinische Studie über den Nystagmus latens, Ophthalmologica **103**:209-224 (April) 1942.

9. Rea, L. R.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938, pp. 65-73.

10. Bielschowsky, A.: Lectures on Motor Anomalies, Hanover, N. H., Dartmouth College Publications, 1943, p. 19.

11. Whitnall, cited by Rea,[9] p. 117.

12. Duane, A.: The Associated Movements of the Eyes, Their Nerve Centers, Conducting Paths, Production, Varieties and Derangements, Am. J. Ophth. **3**:16-26 (Jan.) 1924.

13. Sherrington, cited by Duke-Elder,[2] p. 579.

nism, its workings and the impulses coming from it to the common motor pathways may be influenced quantitatively and qualitatively for smooth or perverted ocular movements by any one or more normal or abnormal impulses reaching it from any direction. Failure to recognize this may give rise to various concepts of the etiology and mechanism of latent nystagmus, such as disturbance of the coordinating centers (Fuchs [5] and Fromaget [7]), inhibition of convergence (Roelofs [14]), labyrinthine disturbance and difference in light tonus (Ohm [4]), congenital lability of the optical apparatus with disturbance of the balance between the right-sided and the left-sided irritation in the supranuclear centers (Verhage [8]) and presence or absence of sharp images on the macula lutea (Kestenbaum [1]).

REPORT OF A CASE

A man aged 24, a Filipino veteran of Bataan, was first seen on Aug. 12, 1942, with complaints of blurring of vision, headache and dizziness. About eight months before, while in action, he sustained an injury to the left occipitoparietal area from fragments of an artillery shell. He was unconscious for many hours and lost much blood. Since a week after he was injured he had noted difficulty in fixing his gaze on objects, which seemed to him to move jerkily from side to side, accompanied with horizontal movements of both eyes, especially the right. On persisting in fixating, he experienced a sensation of instability and frontal, and sometimes occipitoparietal, headache. There was no rotatory vertigo or deafness. He had had no ocular complaints before this head injury. He had malaria and dysentery while a prisoner of war in Capas, but no venereal infection. There had been slight subjective improvement since the onset of his complaints.

He appeared fairly well nourished. There were a thick linear scar, about 4 inches (10 cm.) long, in the left occipitoparietal region and a superficial scar back of the left pinna. There was slight intermittent exotropia, with the right eye usually deviating; this disappeared, especially on fixation for near objects. With the eyes in the primary position for far vision there was no definite gross nystagmus. With the eyes right, spontaneous, transient, jerky horizontal nystagmus of both eyes to the right was present. With eyes left, a similar nystagmus, of lesser intensity, was noted. When the eyes were in the primary position and the left eye was covered, pronounced jerky nystagmus of both eyes would appear, which increased in amplitude and frequency as the eyes were turned to the right but disappeared as the gaze was turned to the left. If the right eye was covered, a similar, less intense, nystagmus appeared, but only on looking to the left. In the dark room, pronounced nystagmus could be elicited by directing the ophthalmoscopic light on the right macula, but not by directing the beam on the other regions of the fundus of the right eye or on the macula of the left eye. No limitation of movement, diplopia or vertical nystagmus was present.

The pupils showed no abnormality. The near point of convergence was 10.5 cm. Far vision was 20/60 in the right eye, with the head turned slightly to the right; it was 20/30 in the left eye and was 20/25 with a correction of 0.62 D. sph. With both eyes fixing, far vision was 20/25, and near vision was ability to read Jaeger type 1. Accommodation was 8.00 D. in the right eye and 8.50 in the left eye. The patient could not sustain reading after a few minutes. Binocular vision

14. Roelofs, cited by Duke-Elder.[2]

was present, as shown by bar reading, the Cruise stereoscope and the Lancaster red-green test. Third degree fusion was present. Its amplitude was not taken. Except for slight pallor of the disk, the fundus was normal on each side. Examination of the visual fields (Ferree-Rand perimeter) showed slight contraction in the left eye, while the field in the right eye could not be accurately taken. With the stereocampimeter the right field was equal to the left, without any hemianopsia. The blindspot of the left eye was normal, while that of the right eye could be mapped out only inaccurately. No scotoma was present on either side.

Hearing in the tuning fork test was practically normal on both sides. Vestibular tests for the horizontal canals gave the following results: Nystagmus was produced in twenty-two seconds when the cold caloric test was performed on the left side and only after thirty-two seconds when it was done on the right side, but in the turning test there was longer after-nystagmus on turning to the left (sixty-two seconds) than on turning to the right (forty-two seconds). The Romberg test was performed with slight swaying. There was no spontaneous past pointing.

The neurologic impression reported (Dr. M. Limson) was purpuric, posttraumatic hemorrhages, meningeal, cortical and intracerebral, especially in the fronto-occipital region and the mesencephalon. Moderate malarial splenomegaly was present. The blood showed malarial parasites but was otherwise normal. The blood pressure was normal. A roentgenogram of the skull showed no fracture. The Wassermann reaction of the blood was 2 plus. The spinal fluid was under normal pressure, with normal cytologic and chemical constituents.

The patient was followed for over six months; he showed slight improvement in headache, dizziness and reading ability, but objectively the latent nystagmus was practically unchanged.

COMMENT

A single 2 plus Wassermann reaction of the blood with a normal spinal fluid, a history of malarial infection and a negative history for venereal disease is of no account. Although the hearing was normal, the caloric test showed vestibular hyposensitivity, but the rotatory test revealed a stronger or longer state of irritability on the right side. This is peculiar, indicating a perverted reaction or an unstable state of the vestibular nuclei (Deiters), and may influence both the production of the transient end nystagmus with both eyes open and the character of the nystagmus with one eye closed. The lower coordinating centers appear to be in the region of Deiters nucleus, in the mesencephalon. But a far stronger influence for the production of the latent nystagmus in this case was the perverted state and function of the higher involuntary ocular motor centers, especially the left occipital lobe, accounting for the appearance of nystagmus when one eye, especially the left, was closed during reflex stimulation by monocular images or strong light. A similar state of unbalanced dysfunction of the voluntary ocular motor centers in the frontal cortex, especially the left, may help explain the difference in intensities of the latent nystagmus with the eyes in the primary position and that elicited with the lateral directions of gaze.

The thesis of this communication is that the peripheral sensory stimuli and the state of stability of the lower coordinating centers, as

well as the balance of the innervating influence of the higher voluntary and involuntary centers, must be considered as a whole in the explanation of the mechanism of latent nystagmus, and probably of all nystagmus. In the case reported, this perverted condition of the higher and lower centers seemed to have followed severe concussion of the brain in a constitution debilitated by malaria, malnutrition and nervous exhaustion brought about by the war. There may, however, have been a congenital or inherent lability of the central nervous system, as in most cases of latent nystagmus reported.

TREATMENT OF CORNEAL ULCER WITH BLOOD AND BLOOD PLASMA

ERWIN E. GROSSMANN, M.D., MILWAUKEE

IT IS notable that the cornea is avascular and derives its nourishment by dialysis from the perilimbal plexus. For this reason healing of corneal infections may become sluggish and prolonged. It is also notable that frequently lesions of the cornea clear up only after vascularization has occurred. According to Duke-Elder,[1] the neovascularization is a response to a call for help by a tissue in difficulty.

In view of the lack of an adequate nutrient supply system in the cornea, it would appear logical to apply the needed nutrient elements directly to the cornea in the form of drops, made up either of whole blood or of blood plasma. Although, so far as is known, this procedure has never been reported as an adjuvant in the treatment of corneal ulcer, excellent results have been reported by other workers from its use in local treatment of ulcers of the leg.

Murray and Shaar [2] reported gratifying results when blood was used in paste form in several cases of indolent decubitus ulcers and in infected wounds. Moorhead and Unger [3] and his associates reported similar excellent results using a red blood cell concentrate.

Naide [4] used blood and concentrated blood plasma with good results in the treatment of ulcers of the leg.

Many ophthalmologists will attest to the fact that in spite of the advent of sulfonamide drugs and other antibiotics many corneal diseases still resist either one or both of these excellent remedies. I have seen instances of epithelial erosion in degenerated phthisical eyes. I sincerely

1. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 3, p. 1840.

2. Murray, C. K., and Shaar, C. M.: Red Blood Cell Paste in the Treatment of Ulcers and Chronically Infected Wounds, J. A. M. A. **125**:779-782 (July 15) 1944.

3. Moorhead, J. J., and Unger, L. J.: Human Red Cell Concentrate for Surgical Dressings, Am. J. Surg. **59**:104-105 (Jan.) 1943.

4. Naide, M.: Treatment of Leg Ulcers with Blood and Concentrated Plasma, Am. J. M. Sc. **205**:489-493 (April) 1943.

doubt whether these recurrent staining lesions are ever manifestations of bacterial disease. These erosions are more likely due to an impoverished corneal tissue, made less permeable to dialysis by the degenerated tissue.

An attempt was made to evaluate the use of whole blood and blood plasma in the treatment of corneal diseases. Six cases are briefly described.

REPORT OF CASES

CASE 1.—A man aged 23 brushed against a twig in the dark, resulting in an epithelial abrasion, measuring 3 mm., of the right cornea. Treatment consisted of atropinization and application of hot compresses and sulfathiazole ointment. After four days of this regimen, the lesion still stained well, the globe remained injected and the lesion showed beginning leukocytic infiltration. Pooled human blood plasma was then used in the form of drops every three hours. In forty-eight hours the lesion failed to stain. Recovery was complete without scarring.

CASE 2.—The patient, aged 24, also received an epithelial abrasion of the cornea from walking into a low-hanging branch in the dark. When, at the end of four days, the customary treatment had failed, blood plasma was used, as in the preceding case. Healing occurred in forty-eight hours.

CASE 3.—A man aged 28 had a lesion of the left eye diagnosed as dendritic keratitis. Treatment consisted of daily applications of half-strength tincture of iodine, atropinization and heat. At the end of ten days the lesion appeared to be spreading. In this case blood was drawn from the antecubital vein and dropped immediately directly onto the cornea. A bandage was then applied. This procedure was repeated daily for six days, when only slight staining could be noted. There was no further spread of the lesion.

CASE 4.—A man aged 30 was seen on several occasions because of recurrent ulcerations in a blind eye manifesting bandlike keratitis. The lesions appeared in dotlike fashion, with frequent coalescence, on the surface of the degenerated cornea. Penicillin and sulfathiazole applied locally failed to remedy the situation. Whole blood was again used, as in case 3, and healing occurred within four days.

CASE 5.—A man aged 52, known to be alcoholic, had an area of denudation involving the lower half of the cornea. The upper border of the lesion showed a yellowish gray infiltration. The general appearance was that of beginning serpiginous ulcer. Whole blood was dropped daily onto the eye, which was then bandaged. Within seven days the lesion healed, leaving no opacities.

CASE 6.—A soldier, aged 42, received a trivial abrasion of the cornea while on duty in England. According to the report by his attending physician (I did not see the patient),[5] the lesion developed into a serpiginous ulcer and became intractable to all forms of treatment. Whole blood was used as a local application several times a day, in addition to the usual routine of atropinization, application of heat, fever therapy, and administration of sulfathiazole and penicillin. The lesion began to show signs of improvement within seventy-two hours, and complete recovery ensued.

CONCLUSIONS

It is obvious that these 6 cases hardly substantiate a definite conclusion regarding the value of blood in the treatment of corneal ulcer. It

5. Shure, I.: Personal communication to the author.

would seem, however, that its use is physiologically sound, and beneficial results have been obtained. The small series of cases does not permit one to choose between the use of whole blood and that of blood plasma. It would appear, however, that the whole blood is preferable. For one thing, whole blood is always conveniently available from the patient. Furthermore, when whole blood is dropped slowly in single drops on the eye, it tends to collect and partly coagulate in the fornices, so that prolonged contact is assured. The application of a bandage further enhances the prolongation of contact.

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News and Notes

EDITED BY DR. W. L. BENEDICT

UNIVERSITY NEWS

Postgraduate Course in Ophthalmology, Medical Extension, University of California Medical School.—Announcement is made of the postgraduate course in ophthalmology to be given by the University of California Medical School, Medical Extension, Sept. 15 to 19, inclusive, 1947, daily from 8:30 a.m. to 12 noon and from 1:30 to 5 p.m.

Requests for information and for registration are to be addressed to Dr. Stacy R. Mettier, head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22.

Graduate Course in Ophthalmology of the University of Rochester School of Medicine.—The fifteenth summer graduate course in ophthalmology of the University of Rochester School of Medicine will be held in Rochester, N. Y., on July 28, 29, 30 and 31, 1947. The program will be as follows:

July 28: "Nystagmus, a Clinical Classification," C. Wilbur Rucker, M.D.; "Oblique Muscle Surgery," John M. McLean, M.D.; "Use of Vasodilators in Ophthalmology," Walter F. Duggan, M.D.; "Lesions of the Optic Chiasm," C. Wilbur Rucker, M.D.; "The Cataract Operation," John M. McLean, M.D.; "Tangent Screen Scotometry," Walter F. Duggan, M.D.; "Classifications of Systemic Hypertensive Vascular Diseases," F. L. Philip Koch, M.D.

July 29: "Misconceptions in Neuro-Ophthalmology," P. J. Leinfelder, M.D.; "Early Glaucoma," Hendrie W. Grant, M.D.; "Slit Lamp Biomicroscopy," Milton L. Berliner, M.D.; "Etiologic Factors in Retinal Hemorrhages," P. J. Leinfelder, M.D.; "Divergent Strabismus," Hendrie W. Grant, M.D.

July 30: "Diisopropyl Fluorophosphate, a New Miotic," Edwin B. Dunphy, M.D.; "Ocular Therapeutics in the Office," Parker Heath, M.D.; "Orthoptics," Le Grand H. Hardy, M.D.; "Headaches and Ocular Pain," Edwin B. Dunphy, M.D.; "Color Vision Tests," Le Grand H. Hardy, M.D.; "Ocular Therapeutics in the Hospital," Parker Heath, M.D.

July 31: Visits to the surgical clinic, Strong Memorial Hospital, and the Bausch & Lomb factory.

Applications should be made to John F. Gipner, M.D., Strong Memorial Hospital, Rochester 7, N. Y. The registration fee is $15; the matriculation fee, $25.

SOCIETY NEWS

Wilmer Residents Association—The sixth clinical meeting of the Wilmer Residents Association was held at the Wilmer Ophthalmological Institute, the Johns Hopkins Hospital and University, on April 17 to 19, 1947. It was a successful meeting, with an attendance of 235.

The meeting began with a joint conference in conjunction with the department of medicine on "Sarcoidosis," led by Dr. McGee Harvey, Dr. A. Murray Fisher and Dr. Alan C. Woods, and "The Progress of Arteriolar Disease," led by Dr. James Bordley and Dr. Jonas S. Friedenwald. The purely ophthalmologic papers then followed: "Ocular Effects of Tridione," Dr. Louise Sloan; "Retrolental Fibroplasia," Dr. William C. Owens; "Roentgen Irradiation in the Treatment of Ocular Diseases Characterized by New-Formed Blood Vessels," Dr. Jack S. Guyton and Dr. Algernon B. Reese; "Experimental Study of Corneal Transplants," Dr. Alfred E. Maumenee; "Surgical Treatment of Paretic Inferior Oblique," Dr. John M. McLean; "Conference on Clinical Pathology," Dr. Russell T. Snip and Dr. Jonas S. Friedenwald; "Cystic Malignant Melanoma of Uvea," Dr. Robert E. Kennedy; "Role of Rutin and Anticoagulants (Dicumarol) in Treatment of Retinal Vascular Disease," Dr. Angus L. MacLean; "Tantalum Implant for Glaucoma," Dr. Malcolm W. Bick; "A Fixation Light for Testing the Six Cardinal Positions of the Eye," Dr. William C. Owens; "Studies of the Physiology, Biochemistry and Cytopathology of the Cornea in Relation to Injury by Mustard Gas [dichloroethyl sulfide] and Allied Agents," Dr. Jonas S. Friedenwald; "Use of Anterior Chamber of the Eye in Cancer Research," Dr. Albert C. Snell Jr. and Dr. John Schilling; "Chemotherapy in Experimental Ocular Tuberculosis," Dr. Alan C. Woods; "Injection of Saline Solution into the Eye in Operations for Retinal Detachment," Dr. Jack S. Guyton; "Sympathetic Ophthalmia," Dr. Samuel D. McPherson; "Demonstration of a New Type Beta Ray Applicator," Dr. Charles E. Iliff; "Experimental Production of Dinitrophenol Cataract," Dr. Wilhelm F. Buschke; "New Types of Plastic Implants After Enucleation," Dr. Russell T. Snip; "The Oculocardiac Syndrome: Differential Diagnosis," Dr. Frank B. Walsh; "Use of Diisopropyl Fluorophosphate (D.F.P.) for Glaucoma," Dr. William G. Marr; "Use of Furmethide [Furfuryl Trimethyl] for Glaucoma," Dr. Ella Uhler Owens; "Apparatus for Chromatic Stimulation of Single Nerve Fibers of Mammalian Retina: Demonstration of Corneal Potential," Dr. Samuel A. Talbot and Dr. Stephen W. Kuffler.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society.—The Louisiana-Mississippi Ophthalmological & Otolaryngological Society held its annual meeting at the Buena Vista Hotel, Biloxi, Miss., on May 5, 1947.

Dr. George Adkins, of Jackson, Miss., president of the society, presided over the meetings. The following papers were presented: "The Rhinoplastic Operation and Restoration of Nasal Function," Dr. Samuel Fomon, New York; "Newer Trends in Treatment of Ocular Diseases," Dr. Peter C. Kronfeld, Chicago; "Ménière's Disease," Dr. Henry L. Williams, Rochester, Minn., and "Allergic Problems Seen by the Ophthalmologist and Otolaryngologist," Dr. Ralph Bowen, Houston, Texas.

Dr. Noel Simmonds, of Alexandria, La., was elected president, and Dr. Edley H. Jones, of Vicksburg, Miss., was reelected secretary. The 1948 Convention will be held in New Orleans.

Central Illinois Society of Ophthalmology and Otolaryngology.—The seventh meeting of the Central Illinois Society of Ophthalmology and Otolaryngology was held at the Hotel Lincoln-Douglas, Quincy, Ill., on April 26 and 27. The following ophthalmologic papers were presented: "Roentgen Therapy of Inflammatory Lesions About the Eye," Edward C. Albers, M.D., Champaign, Ill.; "Narrow Angle Glaucoma," "The Filtering Scar" and "Preparation and Preservation of Ophthalmic Solutions and Ointments," Peter C. Kronfeld, M.D., Chicago (by invitation).

Association for Research in Ophthalmology.—The sixteenth scientific meeting of the Association for Research in Ophthalmology was held at Atlantic City, N. J., on June 10, 1947. The following papers were presented: "Bilateral Granulomatous Uveitis from the Use of Horse Serum in Rabbits," T. F. Schlaegel Jr., M.D., Indianapolis; "Stimulation of Corneal Epitheliation with Local Application of Erythrocytes," Frank W. Newell, M.D., Chicago; "Effect of Diisopropyl Fluorophosphate on the Capillaries of the Anterior Segment of the Eye," Ludwig von Sallmann, M.D., New York; "Transfer of Ascorbic Acid and Related Compounds Across the Blood-Aqueous Barrier," V. Everett Kinsey, Ph.D., Boston; "Acute Reversible Cataract in Chicken Due to Various Nitrocompounds," Wilhelm Buschke, M.D., Baltimore; "Virus Studies in Lymphomatoid Disease of the Ocular Adnexa," Alson E. Braley, M.D., and Rose Alexander, M.S., New York; "Cyanide Inhibition of Corneal Respiration," W. A. Robbie, Ph.D.; P. J. Leinfelder, M.D., and T. D. Duane, M.D., Iowa City; "Ocular Effects of Trimethadione (Tridione)," Louise L. Sloan, Ph.D., and Anita Peck Gilger, M.D., Baltimore; "Reaction of Various Types of Fat Transplanted into the Orbit of Guinea Pigs Prior to the Development of Exophthalmos," George K. Smelzer, Ph.D., New York; "Conjunctivitis with Membrane Formation," Michael J. Hogan, M.D., San Francisco; "Studies of the Physiology of the Eye Using Tracer Substances," Walter S. Wilde, Ph.D.; Roy S. Scholtz, M.D., and Dean B. Cowie, Ph.D., Baltimore; "Pityrosporum Ovale in Seborrheic Blepharitis and Conjunctivitis," J. S. Gots, M.D., Philadelphia, Phillips Thygeson, M.D., San Francisco, and M. Waisman, M.D., Tampa, Fla.

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom was held in the department of zoology of the University of Glasgow, Scotland, on Thursday, Friday and Saturday, March 27, 28 and 29, 1947. The president was Dr. A. J. Ballantyne.

The following papers were read: "Induction of an Experimental Tumor of the Lens," Prof. Ida Mann; "An Unusual Form of Retinal Detachment (Cystic?) Seen in Children," Mr. F. A. Juler; "A Classification of Epiphora, with Comments on Diagnosis and Treatment," Dr. H. M. Traquair; "Some Aspects of Lid and Socket Repair," Mr. J. Scott Tough (introduced by the president); "Bilateral Retinal Detachment Associated with Choroidal Cyst: A Clinical and Pathologic Report," Dr. J. Pendleton White and Dr. I. C. Michaelson; "Corneal Blood Staining," Prof. A. Loewenstein; "Reiter's Disease," Mr. R.

Lindsay-Rea; "Relation of Sjögren's Disease, Plummer-Vinson Syndrome and Ariboflavinosis," Dr. E. Godteredsen; "Holes at the Optic Disk," Mr. H. Neame; "Self-Inflicted Ocular Injuries," Dr. L. B. Somerville-Large; "Vision During Glancing Movements of the Eyes," Dr. G. H. Bell and Dr. J. B. de V. Weir (introduced by Prof. W. J. B. Riddell); "Vascular Disturbances in the Eye Following Concussion Injuries," Dr. A. M. Wright Thomson; "Ocular Infections with Cysticercus cellulosae," Prof. W. H. Melanowski; "Schnabel's Cavernous Atrophy," Mr. Eugene Wolff, and "Some Aspects of Disease Affecting the Retinal Veins," Dr. A. J. Ballantyne and Dr. I. C. Michaelson. The address of the president was entitled "De Senectute."

The main topic consisted in a discussion of "Rhinology in Relation to Ophthalmology." Dr. J. Marshall, Mr. G. H. Howells and Dr. R. McWhirter opened the discussion.

On one afternoon there was a clinical meeting at the Glasgow Eye Infirmary. The annual dinner took place in the Hall of the Royal Faculty of Physicians and Surgeons on Thursday, March 27, and the other entertainments consisted in visits to a steel mill, to the Glasgow Art Galleries and to the Hunterian Museum.

GENERAL NEWS

Dr. Parker Heath Appointed Pathologist at Massachusetts Eye and Ear Infirmary.—Dr. Parker Heath, of Detroit, on July 1, 1947 will become pathologist at the Massachusetts Eye and Ear Infirmary, Boston. He will also be in charge of postgraduate teaching of ophthalmology at Harvard Medical School, with the rank of clinical professor.

Stanford University Eye Bank.—Officials of Stanford University have announced the establishment of an eye bank, to be located at Stanford University Hospitals, San Francisco. The eye bank, which is under the direction of Dr. Dohrmann K. Pischel, will be similar to those now established in New York, Boston and Chicago. A bill now pending will, on passage, make it lawful to will eyes in the state of California.

Institute of Ophthalmology, New York.—The ninth annual meeting of the staff and alumni of the Institute of Ophthalmology, Presbyterian Hospital, New York, was held on May 2 and 3, 1947.

The program was as follows: "Primary Epithelial Tumors of the Ciliary Body," Dr. Joseph Wadsworth, New York; "Achromatopsia: Report of a Case," Dr. Louise Goux, Detroit; "Protein Derangement in Diabetes and its Effect on Diabetic Retinopathy," Dr. G. T. Schwarz, Cleveland; "Virus Diseases of the Cornea," Dr. A. E. Braley, New York; "Melanosis Oculi with Secondary Glaucoma," Dr. F. P. Calhoun Jr., Atlanta, Ga.; "The New Radiotherapy Department," Dr. G. Merriam and Dr. Hayes E. Martin, New York; "Technic of Muscle Surgery" (motion pictures), Dr. Lloyd Morgan, Toronto, Canada; "Pigmented Tumors," Dr. A. G. Reese, New York; "Reaction of Fat Transplantation into Orbit: Regeneration of Corneal Epithelium as Affected by Basal Metabolic Rate," Dr. George K. Smelser, New York; "Surgical Correction of Paresis of the Superior

Oblique Muscle," Dr. W. P. McGuire, Winchester, Va.; "Evaluation of Aniseikonia," Dr. John P. Macnie, New York; "Congenital Impatency of the Nasolacrimal Duct," Dr. Dupont Guerry, Richmond, Va.; "Recent Development in Color Vision Testing," Dr. LeGrand H. Hardy, New York; "Amblyopia from Cinchona Compounds," Dr. H. Rhodes, Pittsburgh; "Split Skin Graft to Cover Dura," Dr. E. B. Hague, Buffalo; DFP [diisopropyl fluorophosphate]," Dr. L. Sallmann, New York; "Brawny Scleritis," Dr. M. Noel Stow, Washington, D. C.; "Choroideremia," Dr. Clement McCullough, Toronto, Canada.

PERSONAL NEWS

Dr. Barkan Guest Speaker at Amsterdam Meeting.—Dr. Otto Barkan is leaving this week for the Netherlands, where he has been invited by the Netherlands Ophthalmological Society and the Society for the Prevention of Blindness to be the guest speaker at their annual meeting in Amsterdam on June 7 and 8, on the subject of "Glaucoma Operations."

Obituaries

WALTER IVAN LILLIE, M.D.

1891—1947

Walter Ivan Lillie was born in Grand Haven, Mich., on Nov. 5, 1891. His untimely death followed acute dilatation of the heart, which was precipitated by his efforts to get his automobile out of a snowdrift while on his way to his office on the morning of Feb. 21, 1947. His death, occurring without any warning to him or to his friends, was a great shock to all who knew him.

He received his degree of Doctor of Medicine from the University of Michigan in 1915 and was appointed a fellow in ophthalmology at the Mayo Clinic in 1917. He served as a member of the Medical Reserve Corps in the American and British Expeditionary Forces in the first World War. He received his degree of Master of Science in ophthalmology at the Mayo Clinic. In 1925 he became a consultant in ophthalmology, and shortly after that he was advanced to associate professor in the Graduate School of the University of Minnesota. In 1933 he was appointed professor of ophthalmology at Temple University School of Medicine, in Philadelphia, in which capacity he continued until his death.

Dr. Lillie was a member of the American Medical Association, the American Academy of Ophthalmology and Oto-Laryngology, the American Ophthalmological Society, the American College of Surgeons, the College of Physicians of Philadelphia, the Harvey Cushing Society, the Sydenham Medical Coterie of Philadelphia, Sigma Xi and Phi Beta Pi medical fraternity. He was a past president of the Section of Ophthalmology of the Philadelphia County Medical Society and chairman of the society's Committee for the Conservation of Vision. He was a guest lecturer in neuro-ophthalmology at the Graduate School of Medicine of the University of Pennsylvania.

Dr. Lillie possessed a charming and gracious personality that endeared him to his friends and colleagues. He had a keen sense of humor and an optimism which never deserted him even in adversity, and which was a source of solace to his friends and patients, who were devoted to him and had unlimited confidence in him. He was especially interested in the problems of the young men in medicine and gave much time to the betterment of his staff, residents and colleagues. He had a sound and brilliant mind and an unusual sense of fairness and honesty.

Dr. Lillie's chief interest was neuro-ophthalmology. His diagnostic acumen, particularly in his field, was unusual. Unfortunately, a text-

book on neuro-ophthalmology which he was writing remains unfinished. He was a resourceful surgeon, who was at his best when the task was especially difficult. He was an outstanding teacher and contributed generously to the ophthalmologic literature.

Dr. Lillie was a remarkably successful departmental head, and he engendered an *esprit d'corps* among his staff, so that all worked enthusi-

WALTER IVAN LILLIE, M.D.
1891-1947

astically, cooperatively and cheerfully together in an atmosphere which was delightful. The devotion of each member of the department to their chief was complete.

The first half of Dr. Lillie's medical career was spent at the Mayo Clinic. While there, he won the friendship, admiration, confidence and respect of the entire staff. He was one of the outstanding consultants at Mayo Clinic. During the last half of his career he reorganized the

Department of Ophthalmology of Temple University School of Medicine and made it outstanding. He was an excellent consultant, always respecting the opinion of the referring ophthalmologist. Dr. Lillie earned an outstanding place in American ophthalmology, where his work was appreciated, his opinion respected and his friendship cherished.

Dr. Lillie's avocation was trap shooting, in which sport he won many trophies. He was twice champion of the Philadelphia Country Club in this sport.

Dr. Lillie was fortunate in having as his life partner Opal Jones Lillie, who was a constant inspiration to him in his lifework. He is survived by his wife and son Robert. A son, Philip, of the Aviation Corps, met his death while in the service of his country in World War II.

In his death, the specialty of ophthalmology has lost one of its most illustrious members, and Philadelphia, one of its most valuable citizens, whose place cannot be adequately filled.

GLEN G. GIBSON, M.D.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ANATOMIC STUDY OF A COMPLEX RETROBULBAR EMBRYONIC MALFORMATION. G. OFFRET, Arch. d'opht. **5:** 51, 1945.

This study was prompted by observations at autopsy. The histologic study presented a complex malformation in the retrobulbar muscle cone characterized by fragments of malpighian epithelium, glands of the skin and glands of the salivary and mucous type. The author discusses tumors of this type occurring in this area according to the classification of Van Duyse: serous cyst, dermoid cyst and teratoma. Excellent photomicrographs accompany the article, and the author states the belief that the tissue which he has studied lies between a dermoid and a teratoma, since the histologic picture revealed such a multiplicity of different tissues.

S. B. MARLOW.

Biochemistry

THE TRYPTOPHAN CONTENT OF THE LENS PROTEINS. H. SÜLLMANN, Ophthalmologica **108:**281 (Dec.) 1944.

The tryptophan contents of the lens proteins of calf, cattle and pig eyes are given. The values were obtained by various methods from hydrolyzed and nonhydrolyzed lens proteins. The author's values are substantially greater than those found in the literature. He considers that most of the tryptophan is found in the beta crystalline form.

F. H. ADLER.

Conjunctiva

CONJUNCTIVITIS ASSOCIATED WITH ERYTHEMA MULTIFORME BULLOSA, E. E. GROSSMANN, Am. J. Ophth. **29:**1146 (Sept.) 1946.

Grossmann reports 7 cases illustrating the association of conjunctivitis with erythema multiforme bullosum. This was apparently checked by sulfathiazole or penicillin therapy, these remedies acting as prophylactic rather than as curative substances.

W. S. REESE.

CONJUNCTIVITIS CAUSED BY VIRUS OF NEWCASTLE DISEASE. J. A. M. A. **132:**169 (Sept. 21) 1946.

In October 1945 in Mikve Israel, a small agricultural settlement, an epidemic of 17 cases of conjunctivitis was reported by J. Yatom. The infection almost exclusively attacked women who had contact with fowl affected with Newcastle disease. No direct spread from man to man was noted, and none of the women infected their husbands. After an incubation period of three days, swelling of the conjunctiva, tarsus and sclera developed without involving the cornea. After an acute stage of three days' duration, characterized by a slightly mucous, purulent secre-

tion, the symptoms subsided within ten to fourteen days, with full recovery. In contrast to the conjunctivitis which is endemic in Palestine, the infection was characterized by being confined to one eye and by not involving the cornea. The epidemic stopped as soon as the affected fowl were slaughtered.

J. A. M. A. (W. ZENTMAYER).

CONJUNCTIVAL FINDINGS IN CASES OF COLD HAEMAGGLUTINATION. S. GÖRDÜREN, Brit. J. Ophth. **30:613** (Oct.) 1946.

Cooling the conjunctival sac was accomplished by using isotonic solution of sodium chloride in varying temperatures. The normal appearance of the blood column in the vessels at 37 C. (98.6 F.) began to disappear at this temperature, and at 27 C. (80.6 F.) the changes became obvious; blood corpuscles agglutinated in the vessels and divided into columns. This took place first in the superficial small vessels; with the decrease in temperature the same process occurred in the deeper vessels, where the blood columns were divided into sections with movements synchronous with systole. When examined with the biomicroscope, the segmented blood columns were irregular and had the appearance of a flocculated mass made of aggregated blood cells. Other changes are described as occurring with variations in temperature.

W. ZENTMAYER.

Cornea and Sclera

PENICILLIN AND VITAMIN C IN THE TREATMENT OF HYPOPYON ULCER. T. C. SUMMERS, Brit. J. Ophth. **30:** 129 (March) 1946.

Summers reports a series of cases of serpent (hypopyon) ulcer. The cases are divided into two classes: those in which the condition was regarded as hopeless from the start, and those in which there was a reasonable expectation of treatment producing a satisfactory result. In the desperate cases treatment was by phenolization of the ulcer, instillation of a solution of penicillin every hour during the day and every three hours during the night, use of atropine and hot pads, short wave diathermy and injection of a preparation of ascorbic acid. In the first group, of 3 cases, the ulcer healed in 1 case, but later a descemetocele developed and the eye was enucleated. In the second group, of 9 cases, no eye was lost. The author is greatly impressed with the value of penicillin. If it is used over a long period the eye remains red, either as the result of the penicillin medication or from irritation due to nonpathogenic organisms. Curettement might be better than phenolization of the ulcer. Penicillin and ascorbic acid together produced a much quicker result than either alone.

A number of colored plates illustrate the article.

W. ZENTMAYER.

CORNEAL COMPLICATIONS FOLLOWING MALARIAL THERAPY. M. CORRADO, Ann. ottal. e clin. ocul. **72:22**, 1946.

Two cases of herpetic keratitis developing during the course of malarial fever therapy are described. The author proposes the administration of massive doses of thiamine hydrochloride in all cases selected for such treatment, purely as a measure for prevention of these corneal lesions.

G. B. BIETTI.

FURTHER OBSERVATIONS ON KERATITIS PRODUCED BY ALCOHOL VAPOR. E. ROMERO ROBLES, Arch. Soc. oftal. hispano-am. **4**: 257 (April) 1945.

This is the continuation of a previous report made in 1944 on this condition. Since then, the author has collected 20 more such cases. There is a characteristic corneal lesion which takes the stain poorly, and there is no clearcut delineation between the affected area and the normal epithelium, this being due to the fact that the damaged epithelium which has separated from the underlying tissues has not yet been shed.

The condition is brought about by exposure to ethyl alcohol vapor, and not directly to the alcohol itself. The symptoms are sudden violent pain, sensation of a foreign body, photophobia, blepharospasm, congestion, lacrimation and involvement of the iris without exudate, all without the presence of a foreign body and with no apparent corneal lesion.

Most cases have occurred in women who have been exposed to alcohol vapor while washing their hair.

The condition is amenable to ordinary treatment and epithelization of the cornea occurs in two, three or four days.

H. F. CARRASQUILLO.

TWO CASES OF PREEPITHELIOMATOUS DYSKERATOSIS OF THE CORNEA AND CONJUNCTIVA (BOWEN'S DISEASE). A. PAULO FILHO and S. R. SÉBAS, Rev. brasil. de cir. **14**: 213 (April) 1945.

The characteristic signs of Bowen's disease are slightly protuberant and vascularized intraepithelial nodules on the cornea, which have a tendency to spread over the surface and are fragile and irregularly shaped. The microscopic aspect is characterized by disorganized texture. The cells are of various sizes; the protoplasm is normal or slightly granular; the nuclei present variations in form and size and abnormal mitosis. In the superficial layer, round cells of dyskeratosis, sometimes surrounded by a membrane, are visible, and histiocytes, lymphocytes and plasma cells are noted in the dermis. Cancerization is always possible. Two cases of Bowen's type of dyskeratosis occurring in men aged 62 and 65, respectively, are presented. Treatment was surgical, and in 1 case there was a recurrence. The treatment indicated is surgical excision, although sometimes total removal of the neoplasm is impossible. In cases of the advanced stage enucleation with removal of the entire bulbar conjunctiva and fornix is indicated. Radium therapy and cauterization are contraindicated.

M. E. ALVARO.

CATARACT DUE TO LOW BLOOD CALCIUM AND ITS MEDICAL TREATMENT. R. WEEKERS, Ophthalmologica **107**: 257 (May-June) 1944.

The author reviews experimental and clinical facts related to the development of cataract associated with low blood calcium. Nine cases are then reported, 7 of which followed thyroidectomy, evidently with inclusion of the parathyroid glands. Two cases followed idiopathic tetany. He believes that in cases following parathyroidectomy cataract

may be prevented by the administration of vitamin D or a preparation known as dihydrotachysterol, together with calcium salts. Early lenticular changes may even be made to regress by this means. F. H. ADLER.

Experimental Pathology

CULTIVATION OF HUMAN TUMOR IN THE ANTERIOR CHAMBER OF THE GUINEA-PIG'S EYE. E. M. BLAKE, Am. J. Ophth. **29:**1098 (Sept.) 1946.

Blake remarks the success of transplants in the anterior chamber over other regions of the body. He describes a case in which fibrosarcoma of the breast was transplanted to the anterior chamber of a guinea pig and calls attention to the possibilities of utilizing the anterior chamber for research problems. W. S. REESE.

ACTION OF NICOTINIC ACID ON THE OPTIC NERVE OF A RABBIT, PREVIOUSLY POISONED WITH QUININE MONOCHLOROHYDRATE. E. MARCONCINI, Arch. oftal. **50:** 19, 1946.

Experiments were conducted to determine the effect of nicotinamide on the optic nerve of a rabbit previously treated with quinine monochlorohydrate.

In the rabbit receiving the quinine monochlorohydrate severe degeneration of the myelin sheath of the optic nerve was noted histologically. In the rabbit treated with nicotinamide (P-P factor) the degenerative changes were insignificant. G. B. BIETTI.

General

OCULAR DECOMPENSATION. E. M. G. GALTON, Brit. J. Ophth. **30:**232 (April) 1946.

In the armed forces large numbers of persons of both sexes were seen who in the ordinary course of events would never have presented themselves for ophthalmic examination. These persons fell into two groups: men and women whose visual acuity was found during routine testing to fall below required service standards, and those with headache as the main symptom. It was only in the minority that an ocular cause for the headache could be substantiated. It was obvious that the majority had been able to do their civilian work quite happily and without difficulty in the presence of refractive errors of all grades, often with some degree of heterophoria as well. It was only when called on to do other types of work, or the same work under less favorable conditions, that some of them experienced symptoms. In other words, ocular symptoms are due not so much to the defects in themselves as to a breakdown in the compensatory factors which exist in even the so-called normal eye. These factors are complex and involve psychologic and mental as well as physical components. In order to emphasize as strongly as possible the dynamic nature of visual symptoms, it is suggested that the terminology of the cardiologist be adopted. The great advantage of the term decompensation is that it implies the possibility of compensation. To

say that a patient has a decompensated exophoria of 12 degrees implies that not only is he having symptoms from his defect but that with treatment his previous state of compensation can be attained. If a patient with low hypermetropia suddenly complains of trouble after near work, he has decompensated hypermetropia and can in the majority of cases be treated without glasses.

W. ZENTMAYER.

General Diseases

REPORT OF A CASE OF IRIDOCYCLITIS ASSOCIATED WITH CHICKENPOX. J. W. HALLETT, Am. J. Ophth. **29:** 459 (April) 1946.

Hallett reports a case of iridocyclitis in a 7 year old boy three days after he contracted chickenpox. Although the attack was severe, the eye recovered fully.

W. S. REESE.

EPIDEMIC KERATO-CONJUNCTIVITIS ASSOCIATED WITH SKIN LESIONS. W. J. O'DONOVAN and I. C. MICHAELSON, Brit. J. Ophth. **30:** 193 (April) 1946.

The cases reported occurred in a general hospital in Egypt during the year 1944-1945.

In 33 of the 66 cases of keratoconjunctivitis there was a concurrent disease of the skin. In 18 of the 33 cases a seborrheic dermatitis was present. In all cases the scalp or face was affected. There was a strong tendency for the cutaneous and ocular lesions to be ipsilateral. In the majority of the cases the onset of the cutaneous lesion preceded that of the ocular one by a definite, but short, interval. A comparison of 33 cases of epidemic virus keratoconjunctivitis without lesions of the skin and the 18 cases with such lesions suggests that the keratoconjunctivitis in the cases with cutaneous lesions was clinically indistinguishable from epidemic virus keratoconjunctivitis.

Lesions of the skin of the face or scalp, especially those of a seborrheic nature, are associated in some cases with keratoconjunctivitis which, on clinical grounds, appears identical with that described as epidemic virus keratoconjunctivitis. The possible analogous nature of the cutaneous lesions of herpes simplex and herpes zoster and in certain cases of epidemic keratoconjunctivitis has been suggested, though by no means proved.

W. ZENTMAYER.

THE RELATION OF CORNEAL VASCULARISATION AND CONJUNCTIVAL TRANSPARENCY TO GENERAL DISEASE. P. A. GARDINER, Brit. J. Ophth. **30:**225 (April) 1946.

This study is based on the examination of 190 unselected members of an R. A. F. hospital staff. A second group was examined which consisted of 150 service personnel who were ill. The diseases were all of a chronic nature. The results obtained from this investigation indicate that examination of the conjunctiva and cornea with a slit lamp provides a clue to the amount of trivial sickness in a group of people over a period varying from three months to two years before examination and also to the amount of sickness they are likely to have in the three months after examination. It is possible to say that persons with a high degree of

corneal vascularity are as a group more likely to report sick than those with a low degree of vascularity. The observations on the conjunctiva must be considered with more reserve, but persons with a high transparency reported sick nearly twice as often before examination as those with a low transparency. The degree of conjunctival transparency and the degree of superficial vascularization of the cornea are shown to be higher in chronically ill persons than in healthy persons. The degree of superficial vascularization of the cornea and the degree of transparency of the conjunctiva are shown to have a relation to the incidence of sickness in a group of healthy people. W. ZENTMAYER.

OCULAR CHANGES IN A CASE OF RIBOFLAVIN DEFICIENCY. G. BASSI and S. JONA, Boll. d'ocul. **24: 3**, 1945.

A 12 year old boy presented the following ocular changes: (1) superficial corneal vascularization, (2) pericorneal injection, (3) clouded aqueous and (4) lacrimation and photophobia. After the daily administration of riboflavin the ocular lesions disappeared completely, only to recur when treatment was discontinued. G. B. BIETTI.

THE RETICULOENDOTHELIAL SYSTEM IN OPHTHALMOLOGY. V. ROSSI, Arch. oftal. **50: 1**, 1946.

The author calls attention to the abundance of reticuloendothelial tissue in the eye, with special reference to its presence in the uveal tract. He briefly reviews the ocular manifestations associated with disorders of reticuloendothelial tissue, the most typical of which are those seen with chronic articular rheumatism, Heerfordt's syndrome (subchronic uveoparotid fever), Niemann-Pick disease and Schüller-Christian disease.

He describes a case of mycosis fungoides (malignant reticular metaplasia) with chorioretinal changes. The patient, a man aged 55, presented a papuloerythematous eruption of the skin of the lid, conjunctival chemosis and tumefaction of the preauricular node, with papilledema, retinal edema, retinal hemorrhages and dilated and tortuous retinal veins. G. B. BIETTI.

Hygiene, Sociology, Education and History

THE OPHTHALMOLOGIST AND THE LAWS CONCERNING MILITARY PENSIONS. JEAN VOISIN, Arch. d'opht. **5:332**, 1945.

This article is a review of the French laws in regard to military pensions and indemnities. The laws which have been passed in France from 1887 up to the present are reviewed. The author points out the importance of the ophthalmologist in the handling of cases involving pensions and in consultation in regard to their authorization. S. B. MARLOW.

Injuries

AN OPERATION FOR POSTERIOR ROUTE EXTRACTION OF INTRAOCULAR FOREIGN BODIES. L. B. SOMERVILLE-LARGE, Brit. J. Ophth. **30:** 208 (April) 1946.

The operation is described in detail. The essential features are summarized as follows: An ample conjunctival flap is cut from before

backward and the episclera completely removed from the trephine site. The sclera over the area that is estimated as nearest the foreign body is trephined with a 1.5 mm. trephine. The base of the disk is dissected from the choroid. The choroid is then coagulated through the trephine hole, with the sharp point of the diathermy apparatus. The tip of the 1.5 mm. point of the magnet is made to enter the hole. The following advantages are claimed for the trephine operation: (1) apposition of a powerful magnet point as near the foreign body as is compatible with safety; (2) production of a minimum choroidal scar; (3) absence of postoperative hemorrhages in the fundus and opacities in the vitreous.

W. ZENTMAYER.

INTRAOCULAR FOREIGN BODY TOLERATED FOR ELEVEN YEARS. B. DA LUZ and R. SÉBAS, Rev. brasil, oftal. **4:** 33 (June) 1946.

Eleven years previously the patient received a penetrating wound from a fragment of steel. A roentgenogram showed a foreign body, which supposedly was removed. For a period of six months preceding this report the eye had been painful. The sequelae of iridocyclitis with iridodonesis and evidence of a peripheral iridectomy were present. The eye was enucleated, and a fragment of oxidized iron was found embedded in the sclera 3 mm. behind the equator of the globe.

M. E. ALVARO.

Lens

ATOPIC CATARACTS. F. C. CORDES and R. CORDERO-MORENO, Am. J. Ophth. **29:**402 (April) 1946.

Four cases of cataract in patients with atopic dermatitis are reported. A brief résumé of the theory of allergy is given, in particular, that of Coca, who coined the word "atopy" because it would seem that cataract formation must be considered an uncommon part of the syndrome. The authors feel that the theory of the atopic shock organ is admissible.

W. S. REESE.

THE RELATIONSHIP OF RUBELLA IN THE MOTHER TO CONGENITAL CATARACTS IN THE CHILD. E. L. GOAR and C. R. POTTS, Am. J. Ophth. **29:**566 (May) 1946.

Goar and Potts report 7 cases of congenital cataract, in all but 1 of which the mother had had rubella. Fair results in all were obtained by the Ziegler through and through discission.

W. S. REESE.

ULTRARAPID OPACIFICATION AND SPONTANEOUS ABSORPTION OF THE CRYSTALLINE LENS. C. ESPILDORA-LUQUE, G. O'REILLY and E. MANNS, Arch. chilena de oftal. **1:**12 (July-Aug.) 1944.

A young man aged 23, who had previously lost the left eye in an accident, had rapid opacification and subsequent absorption of the lens of the right eye. The patient was myopic. He had had a stationary small posterior cortical cataract. Without any apparent cause, the lens became completely opaque in forty-eight hours. This was accompanied with severe pain and congestion of the anterior segment of the eye, but no signs of hypertension or inflammatory reaction were evident. Roent-

genograms of the teeth showed a premolar with a metal filling extending all the way to the apex. This tooth was extracted, and dramatic disappearance of the symptoms and absorption of the cataractous lens took place. The eye returned to normal, and vision was normal with a correction of + 3.50 D.

The authors express the belief that the cataract was brought about by a toxic chemical action from the metal in the filling of the bicuspid.

H. F. CARRASQUILLO.

Neurology

DYSTROPHIA MYOTONICA (STEINERT'S DISEASE): IMPORTANCE OF CATARACT AND DISTURBANCES OF METABOLISM. THERAPEUTIC EFFECTS OF VITAMIN E. A. FRANCESCHETTI and R. S. MACH, Helvet. med. acta **11**: 887, 1944.

Franceschetti and Mach describe 3 personally observed cases of dystrophia myotonica and stress the importance of cataract in the differential diagnosis. In the atypical forms of Steinert's disease only the presence of a cataract permits a definite diagnosis. The biomicroscopic examination of the crystalline lens is indispensable for the differentiation of myotonic dystrophy from other myopathies, particularly from congenital myotonia. The appearance of creatinuria after an aminoacetic acid tolerance test is of great diagnostic value in that in cases of progressive muscular atrophy, congenital myotonia, myasthenia gravis and the muscular atrophies of the Charcot-Marie type creatinuria is nearly always increased after the test, but neither spontaneous nor induced creatinuria is necessarily present in cases of myotonic dystrophy. They observed the favorable effect of vitamin E in a case of myotonic dystrophy, as well as in a case of idiopathic (pseudohypertrophic) muscular dystrophy (Erb's disease). The therapeutic effect of vitamin E on the muscular function is accompanied with improvement in the creatine metabolism. Myotonic dystrophy has a special place in that some of its symptoms indicate a lesion of the endocrine glands, whereas others indicate a nervous origin. It is possible that both these factors are involved.

J. A. M. A. (W. ZENTMAYER).

Operations

THROMBIN TECHNIQUE IN OPHTHALMIC SURGERY. T. G. W. PARRY and G. C. LASZLO, Brit. J. Ophth. **30**: 176 (March) 1946.

Plastic surgeons apply thrombin to the actual surfaces of the graft and bed, and it is generally agreed that the adhesion of such a graft becomes firm enough in many cases to dispense with sutures. It was thought that thrombin might be of greatest value, if successful, in cataract surgery and in conjunctival plastic procedures involving the conjunctiva in general, as in cases of squint.

In cataract operations the section was made in the usual manner with a large conjunctival flap. After completing the removal of the lens, the flap was grasped in two forceps and turned with its inner surface upward. Previously prepared thrombin was instilled on to the turned-up surface and on to the raw area of the globe and the flap placed back and ironed out—in some cases the thrombin was introduced under the flap while in place.

In cases of squint, surgical gut was used for suturing the muscle and thrombin instilled over the whole area of operation; then the edges of the conjunctival wound were immediately brought into apposition and held in this position for approximately one minute.

In a few cases slight crushing together of the edges of the wound with a toothed fixation forceps was carried out.

The hemostatic power of thrombin was also noticeable, and this property alone might well be made further use of in ophthalmic surgery.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

BEHAVIOR OF INTRAOCULAR TENSION FOLLOWING SURGICAL PROCEDURES ON THE ABDOMEN. G. MISSIROLI, Boll. d'ocul. **24:** 137, 1945.

In 1940 Bietti demonstrated the occurrence of a marked fall in intraocular tension following intestinal occlusion. Bolettieri later confirmed these observations experimentally. Missiroli states that ocular hypotony also occurs after various abdominal operations, especially gastric resection, and after intestinal perforation and concludes that there is a definite relationship between the patient's postoperative course and the behavior of the intraocular tension. He believes that this sign has a definite prognostic value in evaluating the course of the surgical condition.

G. B. BIETTI.

Parasites

SCHISTOSOMIASIS OF THE CONJUNCTIVA. G. BADIR, Brit. J. Ophth. **30:** 215 (April) 1946.

Schistosomiasis of the conjunctiva is rare in relation to the large number of patients with schistosomiasis in Egypt. This relative rarity may be either because schistosome infection through the conjunctiva is exceptional or because infection of the conjunctiva with this worm passes unnoticed by the patient until cured with the urinary infection through specific treatment. A case of schistosomiasis occurring in a boy aged 12 years is reported. A tumor had been present in the palpebral conjunctiva of the left upper eyelid near the inner canthus for more than a month. Both eyes showed cicatizing trachoma with pseudocysts (T 3). The swelling was excised. A provisional diagnosis of hypertrophied tarsus was made. The pathologic examination showed a large number of terminal-spined schistosome ova arranged in groups and surrounded by tuberculoid nodules of various sizes. A male and a female schistosome were seen in cross section, lying in a dilated branch of the superior orbital vein. The pathologic report is given in detail. There is a discussion of the way in which the schistosomes reach the conjunctiva and of the possibility that the adult worms may pass from the portal to the systemic circulation through the large anastomotic veins between the two systems.

The article is illustrated.

W. ZENTMAYER.

The Pupil

CLINICAL ASPECTS OF THE PROBLEM OF THE TONIC PUPIL. MARIA ANDREA-VAN LEEUWEN, Ophthalmologica **111:** 339 (June) 1946.

This paper deals chiefly with the question of whether the tonic pupil has a hereditary basis. It was not possible to obtain any evidence

of this, and neither the patients with tonic pupil nor their relatives show any signs of a congenital degenerative disorder of the central nervous system. The families examined, however, showed a definite hereditary basis of allergic and angioneurotic manifestations. Herpes zoster was noted to have occurred frequently among the patients with tonic pupil and their relatives. This peculiar fact, according to the author, justifies a systematic search in further cases of tonic pupil and amongst the patients' families.

F. H. Adler.

Retina and Optic Nerve

Recurrent Edema of the Macula, or Serous Central Retinitis. D. Diaz Dominguez and E. Arques Girona, Arch. Soc. de oftal. hispano-am. **6**: 38 (Jan.) 1946.

The authors describe a condition of the retina under the name recurrent edema of the macula, or serous central retinitis. They believe that the condition is similar to that described by other observers on different occasions (von Graefe, in 1860, and, later, Guist, Gissy, Fritz Rank, Horniker, Asayamsa and others) under various names, such as central recurrent retinitis, preretinal edema, ablatio retinal macularis, capillary spastic retinitis and serous central retinitis.

The condition when reported by von Graefe was supposedly rare, but according to the authors it is seen comparatively frequently. They have observed 14 cases.

The patients complain of a veil in front of the eyes which annoys them; their visual acuity, however, is normal. Ophthalmoscopic study reveals a bright raised circular area in the macula containing disseminated white or yellowish white dots. The condition occurs in adults, is not serious and disappears after a time without any definite treatment. It has, however, a tendency to recur. The etiology of the disease is unknown.

H. F. Carrasquillo.

Therapeutics

The Therapeutic Treatment of Uveitis Associated with Toxoplasmosis. A. C. Krause and L. F. Smith, Am. J. Ophth. **29**: 977 (Aug.) 1946.

Krause and Smith conclude that the sulfonamide compounds with typhoid therapy may have value in the treatment of uveitis associated with toxoplasmosis.

W. S. Reese.

The Use of Crystalline Penicillin in Corneal and Intraocular Infection. F. Juler and G. T. Johnson, Brit. J. Ophth. **30**:204 (April) 1946.

The authors have found the use of crystalline penicillin to have been beneficial in treatment of infective ulceration of the cornea, deep recurrences of keratitis rosacea, infections through the filtration scar of a trephine operation, infections of perforating wounds and in intraocular operations. Sodium penicillinate was used; it is emphasized that the

drug be rubbed well under any advancing edge of ulceration and into any infiltration. No evidence of harm to the tissues has been seen.

W. ZENTMAYER.

OCCUPATIONAL THERAPY IN EYE WARDS. W. O. G. TAYLOR, Brit. J. Ophth. **30**:456 (Aug.) 1946.

Taylor enumerates the following crafts suitable for patients with ocular disorders who need occupational therapy, roughly graded in terms of the amount of vision required, although all can actually be performed by the blind: rug wool weaving, Persian rug knotting, macramé knotting, knitting machine work, basketry, sea grass weaving, weaving on a scarf loom and tablet weaving. The various crafts are illustrated.

W. ZENTMAYER.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

Dec. 16, 1946

INSTRUCTION HOUR

Application of Color Vision in Clinical Practice. DR. GERALD FONDA, Newark, N. J. (by invitation).

A film strip in color, developed at the School of Aviation Medicine, Randolph Field, Texas, was used by courtesy of the School of Aviation Medicine.

The film strip demonstrated the appearance of colored lights and the spectrum to the totally color-blind subject and to the subject with red-green blindness, the attributes of color, classification of color vision deficiencies, methods of administering tests with the pseudoisochromatic plates and use of the School of Aviation Medicine's Color Threshold Tester.

The use of pseudoisochromatic plates was emphasized as a good screening test and that of the Color Threshold Tester as a quantitative color vision test.

There is no cure for congenital color vision deficiencies. An explanation was given of the increase in number of pseudoisochromatic plates read after the so-called cure for color blindness. Reasons were also stated that the Holmgren yarn test is not a good test for color vision.

OBITUARIES

Obituary: Robert K. Lambert, M.D. DR. SAMUEL GARTNER.

This obituary was published in the February issue of the ARCHIVES, page 232.

REPORT OF CASES

Traumatic Prolapse of the Vitreous into the Anterior Chamber: Report of a Case Studied with Fluorescein. DR. GERALD J. HALTON.

Intraocular tension became elevated after trauma to the eye. When the tension would no longer respond to miotics, paracentesis was performed, yielding vitreous, the presence of which in the anterior chamber had not previously been suspected.

As a means of studying the extent of prolapse of the vitreous, fluorescein was given by mouth, and the prolapsed vitreous showed the fluorescein stain in fifteen minutes. Fluorescein was not noted

in the aqueous surrounding the prolapsed vitreous, nor was it observed in the aqueous of the normal eye.

DISCUSSION

DR. WILLIS S. KNIGHTON: First, this case demonstrated how difficult it is to recognize prolapsed vitreous in the anterior chamber with the slit lamp, especially when its presence is not suspected. Second, it showed how much easier it is to see vitreous after disruption by injury, in this case, paracentesis. Third, it demonstrated the value of fluorescein in determining the extent of vitreous in the anterior chamber.

Treatment of Epiphora by Electrocoagulation. DR. DAVID J. MORGENSTERN, Brooklyn (by invitation).

Electrocoagulation is valuable in the treatment of epiphora. Applied to the lacrimal and accessory lacrimal glands, it reduces secretion of tears. Lightly applied at the base of an everted punctum, it produces a cicatrix which draws the punctum backward. Spots of electrocoagulation placed more deeply along a relaxed lower lid tighten the lid, making a firm contact with the eyeball. Conjunctival hypertrophy or tumor causing epiphora can be destroyed. In the intranasal operation for drainage of an infected lacrimal sac, the sac is first entered and partially sterilized by means of an insulated electrode passed through the canaliculus. Similarly applied, electrocoagulation combats threatened closure of the new intranasal opening and reopens such a closure occurring after operative drainage of the sac, as well as corrects obstruction anywhere in the lacrimal sac or along the nasolacrimal duct. Such an obstruction may be a polyp, cyst, scar tissue or bony sclerosis. Blockage at the orifice of the duct may similarly be destroyed. The current used is about 1,000 milliamperes when the active electrode is directly connected with the indifferent electrode. In cases of congenital blockage at the lower end of the nasolacrimal duct channelization can be produced. When excessive folds of mucous membrane are present in the duct and abnormal meatal continuation of the duct downward (often terminating in a minute opening or slit) causes tearing, an adequate opening can be formed by electrocoagulation.

PAPER OF THE EVENING

Tobacco Amblyopia. DR. CHARLES L. SCHEPENS, Brussels, Belgium.

In peacetime there is an unexplained geographic variation in the incidence of tobacco amblyopia.

During the last war it was much more frequent than usual in Belgium. Reasons given are: (1) consumption of wartime tobacco; (2) mental shock, and (3) undernourishment, especially the lack of fats and animal proteins. With the diet poor in these substances, the disease appears in younger patients and causes chronic symptoms of general tobacco poisoning. The intake of large doses of vitamin A, thiamine chloride and riboflavin has not affected the course of the disease in my Belgian patients, who had no signs of vitamin B deficiency. These observations can be reconciled with Dr. Carroll's

views by admitting that vitamin B deficiency, when present, is an important etiologic factor in tobacco amblyopia. When it is not present, however, as in my cases, the intake of vitamin B has no effect on the amblyopia.

Careful study of the blood and of hepatic function in malnutritional as well as in tobacco amblyopia may prove useful.

The defects in the visual fields suggest that the disease begins in the retina and not in the optic nerve. The first campimetric sign is enlargement of the normal angioscotoma. Later, the pericecal maze, or lacunar scotoma, contains nuclei (small areas of absolute scotoma). The cecocentral localization of the tobacco scotoma probably results from the greater sensitivity of the macular cones together with a tendency of the normal angioscotoma to enlarge. The cecocentral area is the only place where these two factors overlap. The probable relation of the cecocentral scotoma to a cilioretinal angioscotoma in many instances is stressed.

At a very early stage the field defects of heredofamilial atrophy of the optic nerve and tobacco amblyopia are similar. They consist in pericecal lacunar scotoma, particularly in the cecocentral region. At a later stage, that of Leber's disease, a pericentral scotoma develops, which is related to the lacunar scotoma. Later still, the blindspot itself enlarges, through atrophy of the optic nerve. The character of these field defects may suggest that heredofamilial optic nerve atrophy begins in the optic nerve and not in the retina.

DISCUSSION

DR. FRANK D. CARROLL: Considering the conditions that prevailed in Belgium from 1939 to 1942, it is remarkable that Dr. Schepens was able to carry on a scientific investigation of the kind he has described. I am glad that we agree on many points. It is rare to see patients with tobacco amblyopia who do not consume at least some alcohol; there is no doubt, however, that there is such a thing as tobacco amblyopia alone. In the literature several hundred cases have been reported. Usher, in a series of 1,100 cases, stated that in over 100 cases the patients were teetotalers. I have seen a small number of patients who did not drink. But in this country most patients with this syndrome do consume alcoholic liquors, sometimes in large amounts. This syndrome can occur in heavy drinkers who do not smoke, but it is rare to find any heavy drinker who does not smoke at least a few cigarets a day.

I agree with Dr. Schepens on the importance of the scotoma rather than of the visual acuity, and his "nasal reading" test is interesting. Patients develop amblyopia during a period of undernourishment or chronic starvation, as Dr. Schepens said. One would not think that in this country, where there is plenty of food, one would see persons with chronic starvation. Certainly, most of the patients with amblyopia do not look malnourished. Alcoholic persons frequently do have evidence of vitamin deficiency; but, as I have stated before, none of my patients with tobacco amblyopia have shown any clinical evidence of vitamin deficiency. The only reason that I was encouraged to

treat them with vitamins was that vitamin therapy seemed to do so much good to patients with alcohol amblyopia, or what might better be called nutritional retrobulbar neuritis.

Dr. Schepens spoke of an ocular disease which was found in persons held as prisoners of war by the Japanese. He pointed out that pericentral scotoma of the type associated with this condition, having a connection with the blindspot, would be rare in persons with tobacco amblyopia. Frequently the vision of these prisoners was 20/70 or 20/100, and in most of them I saw (and I did not see a great many) it did not improve. I do not think that one knows the whole story about this form of amblyopia. One of the patients told the Army physicians that at his camp in the Philippines, where many of the prisoners acquired this condition, they were able to steal some unpolished rice from the Japanese and that over a period of a month or two, while they had this diet of unpolished rice, their vision improved tremendously. When they had to go back to their diet of polished rice, their vision became worse. The only difference between polished and unpolished rice I know of is the thiamine content.

I should like to ask Dr. Schepens what tests should be made to determine "hepatic deficiency." Hepatic function tests are not as satisfactory as one would like.

I agree that this condition has a very fluctuating course and that some patients who are expected to recover rapidly do not do so. A very few patients who stopped the use of alcohol and tobacco and who were given a good diet have not shown improvement in vision. I am sure there are some problems and factors which are not yet understood.

Dr. C. L. Schepens, Brussels, Belgium: In a case of tobacco amblyopia a number of things are to be borne in mind. The first is that tobacco amblyopia is due to tobacco poisoning, and therefore tobacco still remains the most important factor. The second point is that of diet. It appears to me that several dietary factors have to be considered. In my cases the lack of proteins and the caloric deficiency certainly were the main elements. Since there was no vitamin B deficiency, it is surprising that vitamin therapy was ineffective. In Dr. Carroll's cases, on the contrary, there was no caloric deficiency but a vitamin B deficiency existed, as it often does in alcoholic patients. This explains his good results with vitamin therapy. I am under the impression that three dietary factors have an influence on the incidence of tobacco amblyopia: the caloric intake, the animal protein intake and the vitamin B intake.

The third point to be considered in a case of tobacco amblyopia is the possible presence of anemia. An experienced clinician in England told me that in his experience chronic anemia is an etiologic factor in tobacco amblyopia. The fourth point is that of hepatic insufficiency. This is of course a great problem. I am not suggesting that it is wrong not to make any of the various tests for hepatic dysfunction in a case of tobacco amblyopia, but I do not know what test should be made. It is my suggestion that research be carried out on this subject. De Schweinitz observed urobilinuria, and more recently Lundberg noted hyperglycemia, in cases of tobacco amblyopia.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Burton Chance, M.D., *Chairman*

George F. J. Kelly, M.D., *Clerk*

Dec. 19, 1946

Experimental Investigation of the Pathogenicity of Diphtheroids Isolated from the Human Conjunctiva. Dr. Charles Weiss and Mr. M. C. Shevky, San Francisco.

Diphtheroids resembling Corynebacterium xerose and isolated from the human conjunctiva grow well in a menstruum of mucin. When suspended in this medium and inoculated into the anterior chamber of the eyes of albino rabbits, cultures retain their viability for several days, while in saline solution they are rapidly destroyed.

Intraocular injections into rabbits of cultures of diphtheroids suspended in saline solution produce moderate inflammation of the ciliary process. With suspensions in mucin the reaction lasts longer, is much severer and is associated with acute keratitis. The lesions usually regress spontaneously within two or three weeks.

Killed cultures of diphtheroids in saline solution produce mild inflammatory changes in the ciliary process which are seen in histologic sections; none of the lesions are visible grossly. In a menstruum of mucin the inflammatory reaction is severer. Since mucin alone is relatively innocuous, it is suggested, on the basis of these studies and those of others, that it protects bacteria from the digestive action of humoral and cellular proteolytic enzymes and other immunologic defense mechanisms. Living diphtheroids are thus permitted to grow and exert their pathogenic activity.

By means of the recently developed methods of Müller and Miller and Pappenheimer and Johnson, it was possible to demonstrate that when diphtheroids are grown in a medium of very low iron concentration they yield a toxic filtrate which is injurious to the uvea and cornea, but not to the skin or conjunctiva, of albino rabbits.

Studies which had been planned on the specific chemotherapy and serum therapy of diphtheroids were interrupted because of the war emergency.

On the basis of these investigations, it may be concluded that diphtheroids which are present on the normal or the inflamed conjunctiva may be considered as potential pathogens, which can exert serious injury when they find their way into the interior of the eye.

DISCUSSION

Dr. Luther Kauffman: I wonder whether in treating conditions involving the conjunctiva the proper procedure would be to get rid of the mucin. I am reminded of the treatment of vernal conjunctivitis with a solution of monohydrated sodium carbonate which Dr. Lehrfeld presented before this section several years ago.

Does Dr. Weiss know whether other bacteria receive the same protection from mucin that these diphtheroids do?

Dr. Burton Chance: The early death of diphtheroids reminds me of my own experience in the early days of my medical practice, when "diphtheria" was an extremely frequent disease. For two winters

I did volunteer work at the old Municipal Hospital; one week 275 sick persons were inspected. Many patients were there because the bacteriologist, who was among the first in the field at that time, had found what he believed to be the diphtheritic organism in material from the conjunctiva and fauces. A few days after the children were admitted, another examination was made, with negative results; this created more than a "headache," both outside and inside the hospital. The children were interested to know whether or not the diagnosis had been correct. The people outside were complaining because their children had been taken away from home and isolated. Having to have their own toothbrushes and carry out other disinfecting measures were the sources of complaints from the children. There were no signs of the old diphtheria of the conjunctival tissues that had prevailed in earlier times. My teachers would refuse to accept a diagnosis of diphtheria unless there were actual structural and infiltrative changes. Dr. Weiss's statement concerning the evanescence of the diphtheroids, therefore, reminded me of that somewhat confusing experience.

DR. CHARLES WEISS, San Francisco: In answer to Dr. Kauffman's question: In general, mucin on the inflamed conjunctiva is due to bacteria. Of course, it may also be due to chemical irritants. On the basis of our work, it seems reasonable to remove it. I mentioned in the course of my presentation that the virulence of a number of other bacteria is increased by mucin. Among them are the meningococcus, the gonococcus, Vibrio comma (cholerae) and the virus of influenza.

Experiences in Testing Color Vision of Persons with Defective Color Sense. DR. BURTON CHANCE.

Dr. Chance stated that early in life he became aware that some persons were color blind, and ever since he had been interested in color. In recent years defective color vision has become the subject of so much investigation that he ventured to tell something of his own interest in it, and to express his beliefs.

The perception of color by human beings is an intellectual process involving psychologic and physiologic factors operating with the effects of light on the chemical elements in the retina. The average person is able to distinguish all seven colors in the visible spectrum—it is not improbable that animals and birds perceive beyond what mankind can define. There occasionally is found a person whose appreciable "spectrum" is shortened at one end or at both ends, and others to whom the ends of the spectrum are only somewhat indistinct—hence the appellation color blind. A better and kinder name is "achromatopsia." More men than women are affected. The defect is transmitted from one generation to another through females, who may not themselves be known to be affected. In pure cases of color blindness the structures of the eyes are not visibly changed. Disease of the retinochoroid may be accompanied with defective color perception; however, the defect in color vision is usually not comparable to that of the persons with a congenital deficiency. A singular anomaly is that presented by several persons with a unilateral deficiency.

The measurement of color perception has aroused serious consideration in the arts and has presented important problems in transportation,

as well as in the selection of members of the military forces. Many persons notable in history have had deficient color perception. Since this defect was pronounced in John Dalton, the famous English physicist, he gave it deep study and devised various schemes for its testing.

Color vision tests have utilized dyed wools, colored charts and colored lights which are compared or matched with certain standard colors, selected after prolonged investigation. In practice, red and green have been the colors for which vision has most frequently been found to be deficient; hence those colors have been adopted as the basic standard for all tests.

Color perception is an unsolved mystery. Many theories have been proposed, but none solves all the problems—their respective advocates are violent contenders for their hypotheses. Thus far, the theory offered by Thomas Young, and later arrived at by similar means by von Helmholtz, has the least uncertainty. According to their theory, each color excites its own system of retinal fibers to the brain.

Being a congenital defect, color blindness cannot be cured, or even reduced—all schemes proposed are only false promises. Subjects can be trained to distinguish one color from another more fully than was possible before intensive training was begun; yet when standard lights or plates are modified, as by foggy atmosphere or color dilutions, the natural defects become manifest.

All subjects have not identical defects; some lack perception of one or two, others of more, even all seven, colors. Observers, therefore, have classified them according to the number. Some showing only a lessening of the color strength are classed as having anomalous vision for those color classes. Increasing attention to "anomalous" color vision is being carried on today. Persons to whom all colored objects are as though they were black are spoken of as having monochromatic vision. Despite all the interest evinced in the past fifty years, no new fundamental aspects of the problem have been solved; their solution rests with the photochemistry of the retina.

DR. GEORGE F. J. KELLY: Dr. Chance spoke of several cases in which there was normal color perception in one eye and abnormal color perception in the fellow eye. Was there any evidence of pathologic change in the fundus of the abnormal eye?

DR. P. ROBB MCDONALD: I have been interested in Dr. Chance's paper because for part of the time that I was in the service I was working with persons who were engaged in developing tests of color vision.

I notice that he also used the term "defective color vision." This is a much more appropriate term than "color blindness." There are many people who have defective color vision but who are by no means color blind. In setting up tests for color vision there were certain requirements. They were essentially as follows: (1) a "job analysis" to determine what color discriminations were actually required, e. g., of pilots, bombardiers and air crew; (2) evaluation of the tests of color vision that were available; (3) development of new tests; (4) determination of the reproducibility, reliability and validity of the tests that were adopted.

The problem of landing a plane in the daytime was not too difficult because clues other than color were usually employed to determine whether one was about to land on a tilled or on a flat surface. At night the problem was more difficult. The runway markers might be colored lights of low intensity; the landing might be made with glide path indicators, or the only communication between the tower and the pilot might be made by a directional light, such as the "biscuit gun." Men who worked about the airplane also had to make certain color discriminations; for example, the wiring was usually coded by color, and the various fuses had color codes.

What every one wanted was a quantitative test of color vision. One of the closest approaches to this was a lantern developed by Dr. Louise Sloan Rowland. The test was modified from one developed by the Royal Canadian Air Force, which, in turn, was a modification of the Williams lantern. The test was called a color threshold test and consisted in presenting eight different colored lights at eight different intensities of illumination. The minimum intensity was that at which most normal persons called the color correctly. A score of 62 to 64 was considered normal.

We were fortunate in having a considerable number of persons with defective color vision at Randolph Field, and Dr. Sloan was able to test them with various tests of color vision. In all, about six tests were used—the Ishihara, the American Optical Company's pseudo-isochromatic plates, an abridged edition of the same, the Rabkin polychromatic plates, an anomaloscope test and the Color Threshold Tester. Several other tests were employed at one time or another. Either before or after this battery of tests the subjects were given "practical" tests, such as the identification of colored flares actually fired from an airplane, the identification of colored signal lights and the identification of colored codes on wires.

On the basis of the results, the Color Threshold Tester was the closest approximation to a quantitative test. In the future there may be two types of color vision tests: one that can be given in the office to determine whether a subject is normal or not, and one the military may use to determine whether the subject has safe color vision or not.

DR. BURTON CHANCE: In the several instances in which I had seen perfect color vision in one eye and defective color perception on the other there were no visible changes in the fundus. If it is true that the perception of color is generated by the action of light on the retinal pigment, it is more than likely that there was something constitutionally lacking in the color-blind eyes that gave rise to the defect. Therefore, in this consideration, I should not class a case of unilateral color blindness presenting changes in the fundus as a case of true color blindness.

I wish to thank Dr. McDonald for telling us something of what was done in the Army in the last war; I regret that I was afforded no opportunity to witness such studies personally.

Book Reviews

Squint and Convergence: A Study of Di-Ophthalmology. By N. A. Stutterheim. Price, 15s. Pp. 95, with 26 graphs and 15 diagrams. London, England: H. K. Lewis & Co., Ltd., 1946.

This book attempts to give a new theory of the origin of comitant strabismus. The work is difficult to evaluate because it lacks clarity of expression. In his previous book, "Indications for the Kinetic Treatment of the Eyes," published in 1931, the author evolved the concept that "not fusion sense but what I called involuntary convergence, the basic movement of the di-ophthalmos or the bi-unial eye, is the kinetic power for bi-foveal single vision."

He starts with the assumption, which is undoubtedly correct, that the cause of comitant squint is to be found in some supranuclear pathway. From this point on, his reasoning is difficult to follow and he comes to the conclusion that "squint is that condition of human vision (the bi-foveal eye) where convergence and its correlate—namely the bi-fovea—are in abeyance."

One gets the impression from reading the book that the ideas it contains have merit but that they could be better expressed.

The second part of the book deals with therapy. This consists in what the author calls kinetic treatment of reflex convergence with a battery of prisms, based on the physiologic law of facilitation of reflexes.

FRANCIS HEED ADLER.

Monocular Vision Training. By Mildred Smith Evans. Price, $3. Pp. 100, with 28 lessons and 18 drawings. Baltimore: Williams & Wilkins Company, 1946.

The author has arranged a series of graded daily tasks for amblyopic eyes covering a period of approximately one month. These include such stock devices for improving eye-hand coordination as tracing and dot drawing. The text, printed in boldface ranging from 24 to 10 point, embodies directions for the exercises, original stories and questions, provocative of thought as well as visual discrimination.

A space is provided in which to write the time required to complete each lesson. This is a worth while detail, for speed must combine with accuracy in meeting the visual tasks of daily life. It was found that a boy of 7 years with a visual acuity of 6/20 (20/70) in his amblyopic eye required approximately one-half hour to finish each daily task. Mrs. Evans has evidently apportioned her material well.

The book is obviously limited, however, in the amount of material it offers. Unfortunately, permanent normal vision can rarely be restored to an amblyopic eye in a month's time. (The inclusion of several tracing sheets instead of one for each picture would contribute toward giving the training a longer time spread.) There is a further limitation in the text, the reading and interpretation of which would be too difficult

for the average child under 7 years of age. Much of the most effective work with amblyopia is accomplished with children under this age.

The price may seem high on first consideration and undoubtedly makes the purchase of this volume prohibitive for most clinic patients. When compared with the cost of half-hour weekly treatments in a private office, $3.00 is not an excessive outlay for four weeks' daily homework.

Mrs. Evans has made a start in the right direction. More such planned exercises are needed. The American Association of Orthoptic Technicians might well make the assembling of a graded library of amblyopic material one of its major projects.

ELIZABETH K. STARK.

The Eye Manifestations of Internal Diseases. By I. S. Tassman, M.D. Second edition. Pp. 614, with 243 illustrations, 24 in color. St. Louis: C. V. Mosby Company, 1946.

According to the author, this book is designed to serve the needs of the ophthalmologist and all others engaged in general or specialized practice. I suppose that Dr. Tassman has steered this difficult middle course as well as could be expected, but his work suffers from the numerous compromises that have had to be made. For example, several chapters are devoted to a description of the eye and its adnexa, the examination of the patient and the routine tests and equipment therein employed. I believe that much of this could have been omitted—the ophthalmologist does not need such primary instruction, and the general practitioner is unlikely either to perform the simpler procedures or to buy the equipment necessary to carry out the more elaborate examinations.

The second part of the book, beginning at chapter VI, describes the ocular manifestations of systemic diseases. The systemic disease is first described, and the consequent ocular lesions are then taken up in adequate, if not encyclopedic, detail. The headings include "Structural, Congenital and Hereditary Manifestations"; "Infections and Focal Infectious Diseases"; "Tuberculosis, Virus, Fungus, and Parasitic Infections"; "Drug Intoxications"; "Diseases of the Cardiovascular System, Blood, Endocrine Glands, Nervous System, Skin and Bone," and "Disorders of Menstruation, Pregnancy, Metabolism and Nutrition." There is a separate chapter on intracranial tumors.

The illustrations, particularly those in color, are good. Unfortunately, most of the reproductions of fundus photography have not come out well, and future editions would be improved if these photographs could be replaced with drawings.

This book lacks the conciseness of its classic predecessors, notably the works of Knapp and Foster Moore; but its faults are more than balanced by its virtues. I have enjoyed reading it and have already referred to it more than once with profit to myself and my patients. I consider it a useful work and have no hesitation in recommending it.

G. M. BRUCE.

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